

**ABSTRACT
OF
The Proceedings of the Thirty-Fifth
Annual Meeting of the Association
of Life Insurance Medical
Directors of America**

PRINTED FOR PRIVATE CIRCULATION

VOL XI.

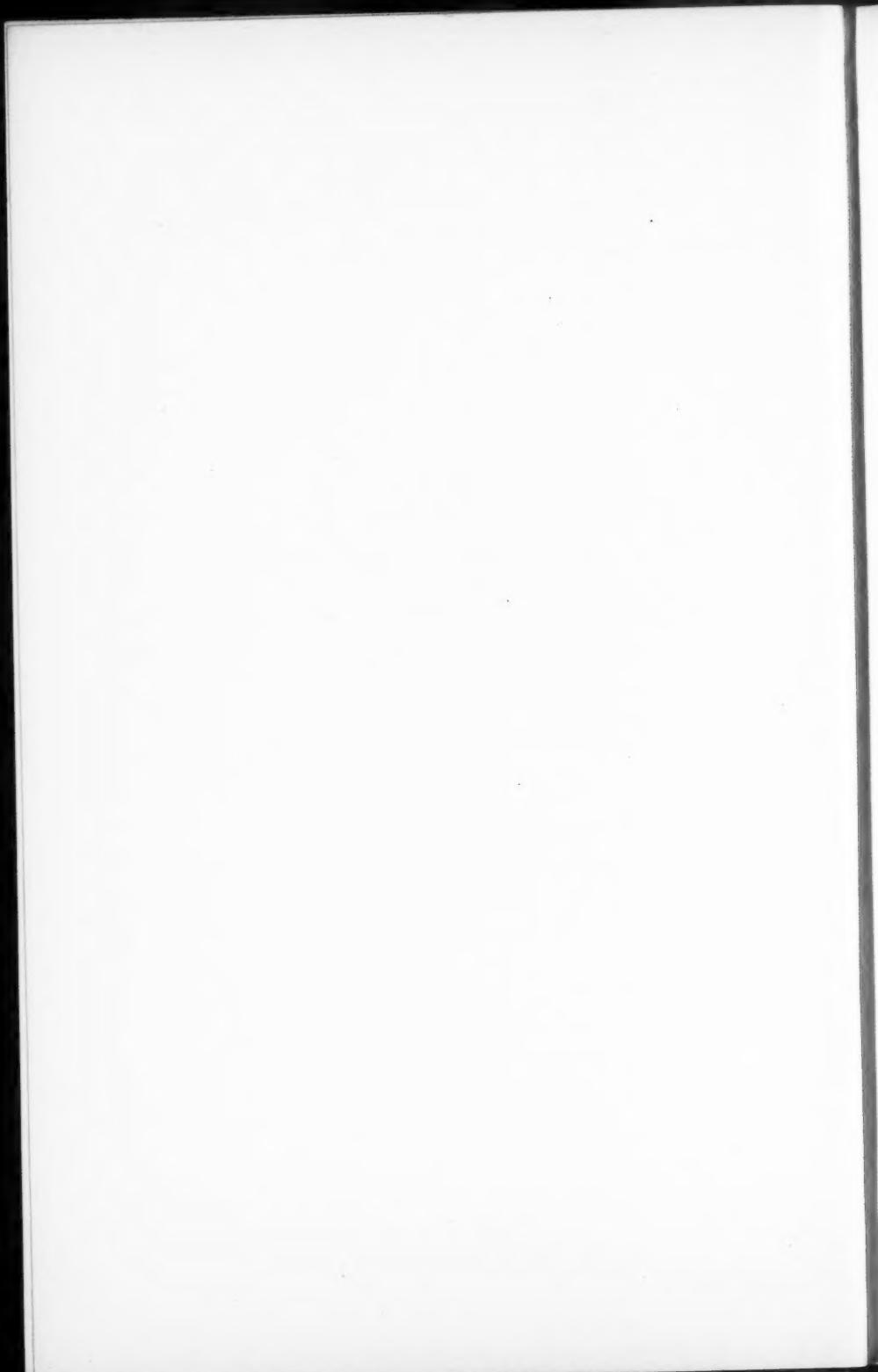
**WALTON, NEW YORK
The Reporter Press
1925**

Copyright, 1925

by

THE ASSOCIATION OF LIFE INSURANCE
MEDICAL DIRECTORS OF AMERICA

**Compiled by the Editor of the Proceedings
by
Order of the Association**



OFFICERS OF THE ASSOCIATION, 1923-1924

WILLIAM R. WARD, M. D. *President*
CHESTER F. S. WHITNEY, M. D. *First Vice-President*
ANGIER B. HOBBS, M. D. *Second Vice-President*
CHESTER T. BROWN, M. D. *Secretary*
CHARLES L. CHRISTIERNIN, M. D. *Treasurer*
EUGENE F. RUSSELL, M. D. *Editor of the Proceedings*

EXECUTIVE COUNCIL

| | |
|---------------------------|--------------------------|
| W. R. WARD, M. D. | T. H. WILLARD, M. D. |
| C. F. S. WHITNEY, M. D. | BRANDRETH SYMONDS, M. D. |
| A. B. HOBBS, M. D. | HARRY TOULMIN, M. D. |
| C. T. BROWN, M. D. | E. K. ROOT, M. D. |
| C. L. CHRISTIERNIN, M. D. | W. E. PORTER, M. D. |
| E. F. RUSSELL, M. D. | HOMER GAGE, M. D. |
| G. A. VAN WAGENEN, M. D. | W. A. JAQUITH, M. D. |
| F. W. DWIGHT, M. D. | T. H. ROCKWELL, M. D. |
| J. ALLEN PATTON, M. D. | F. S. WEISSE, M. D. |
| W. W. BECKETT, M. D. | A. S. KNIGHT, M. D. |
| J. B. STEELE, M. D. | T. F. McMAHON, M. D. |
| J. W. FISHER, M. D. | F. L. GROSVENOR, M. D. |
| O. H. ROGERS, M. D. | |



OFFICERS OF THE ASSOCIATION
SINCE ITS ORGANIZATION

Presidents

| | |
|------------------------------|-----------|
| JOHN M. KEATING, M. D. | 1889-1891 |
| FRANK WELLS, M. D. | 1891-1894 |
| EDGAR HOLDEN, M. D., Ph. D. | 1894-1897 |
| H. CABELL TABB, M. D. | 1897-1899 |
| GEORGE R. SHEPHERD, M. D. | 1899-1902 |
| EDWARD H. HAMILL, M. D. | 1902-1904 |
| JOHN W. FISHER, M. D. | 1904-1905 |
| OSCAR H. ROGERS, M. D. | 1905-1908 |
| THOMAS H. WILLARD, M. D. | 1908-1910 |
| GEORGE WILKINS, M. D. | 1910-1911 |
| BRANDRETH SYMONDS, M. D. | 1911-1912 |
| HARRY TOULMIN, M. D. | 1912-1913 |
| EDWARD K. ROOT, M. D. | 1913-1914 |
| WILLIAM EVELYN PORTER, M. D. | 1914-1915 |
| FRANKLIN C. WELLS, M. D. | 1915-1916 |
| HOMER GAGE, M. D. | 1916-1917 |
| WALTER A. JAQUITH, M. D. | 1917-1918 |
| THOMAS H. ROCKWELL, M. D. | 1918-1919 |
| FANEUIL S. WEISSE, M. D. | 1919-1920 |
| AUGUSTUS S. KNIGHT, M. D. | 1920-1921 |
| THOMAS F. McMAHON, M. D. | 1921-1922 |
| FRANK L. GROSVENOR, M. D. | 1922-1923 |
| WILLIAM R. WARD, M. D. | 1923- |

Vice-Presidents

| | |
|----------------------------|----------------------|
| GURDON W. RUSSELL, M. D. | 1889-1892 |
| LEWIS MCKNIGHT, M. D. | 1898-1892 |
| JOHN W. FISHER, M. D. | 1892-1895, 1902-1904 |
| H. CABELL TABB, M. D. | 1892-1897 |
| FREDERICK W. CHAPIN, M. D. | 1895-1896 |
| JOSEPH H. WEBB, M. D. | 1896-1897, 1898-1899 |
| GEORGE R. SHEPHERD, M. D. | 1897-1899 |
| EDWARD H. HAMILL, M. D. | 1899-1902 |
| THOMAS H. WILLARD, M. D. | 1902-1908 |

viii. Officers of the Association

| | |
|------------------------------|-----------|
| OSCAR H. ROGERS, M. D. | 1904-1906 |
| GEORGE WILKINS, M. D. | 1906-1910 |
| ROBERT L. LOUNSBERRY, M. D. | 1908-1909 |
| GEORGE A. VAN WAGENEN, M. D. | 1909-1910 |
| BRANDRETH SYMONDS, M. D. | 1910-1911 |
| HARRY TOULMIN, M. D. | 1910-1912 |
| EDWARD K. ROOT, M. D. | 1911-1913 |
| WILLIAM EVELYN PORTER, M. D. | 1912-1914 |
| FRANKLIN C. WELLS, M. D. | 1913-1915 |
| HOMER GAGE, M. D. | 1914-1916 |
| WALTER A. JAQUITH, M. D. | 1915-1917 |
| ARTHUR B. WRIGHT, M. D. | 1916-1918 |
| THOMAS H. ROCKWELL, M. D. | 1917-1918 |
| FANEUIL S. WEISSE, M. D. | 1918-1919 |
| AUGUSTUS S. KNIGHT, M. D. | 1918-1920 |
| THOMAS F. McMAHON, M. D. | 1919-1921 |
| FRANK L. GROSVENOR, M. D. | 1920-1922 |
| WILLIAM R. WARD, M. D. | 1921-1923 |
| C. F. S. WHITNEY, M. D. | 1922- |
| ANGIER B. HOBBS, M. D. | 1923- |

Secretaries

| | |
|------------------------------|-----------|
| FRANK WELLS, M. D. | 1889-1891 |
| ELIAS J. MARSH, M. D. | 1891-1894 |
| OSCAR H. ROGERS, M. D. | 1894-1900 |
| BRANDRETH SYMONDS, M. D. | 1900-1907 |
| WILLIAM EVELYN PORTER, M. D. | 1907-1912 |
| FANEUIL S. WEISSE, M. D. | 1912-1918 |
| ANGIER B. HOBBS, M. D. | 1918-1923 |
| CHESTER T. BROWN, M. D. | 1923- |

Treasurers

| | |
|--------------------------------|-----------|
| JOHN W. BRANNAN, M. D. | 1889-1899 |
| FRANK S. GRANT, M. D. | 1899-1903 |
| AUGUSTUS S. KNIGHT, M. D. | 1903-1918 |
| CHARLES L. CHRISTIERNIN, M. D. | 1918- |

Editors of the Proceedings

| | |
|--------------------------|-----------|
| ANGIER B. HOBBS, M. D. | 1917-1918 |
| ROBERT M. DALEY, M. D. | 1918-1923 |
| EUGENE F. RUSSELL, M. D. | 1923- |

An Abstract of the Proceedings
OF THE
Association of
Life Insurance Medical Directors
of America

THIRTY-FIFTH ANNUAL MEETING

The Thirty-fifth Annual Meeting of the Association of Life Insurance Medical Directors of America was held in the Home Office of the Mutual Benefit Life Insurance Company, Newark, N. J., on October 23rd and 24th, 1924. President William R. Ward was in the chair.

The following members and delegates were present at some time during the sessions: E. H. Allen, H. B. Anderson, W. McP. Armstrong, W. B. Aten, W. C. Bailey, H. A. Baker, W. B. Bartlett, J. T. J. Battle, W. W. Beckett, C. D. Bennett, C. C. Birchard, W. B. Blackford, W. M. Bradshaw, B. C. Brooke, C. T. Brown, W. H. Browne, B. F. Byrd, F. H. Carber, L. D. Chapin, C. L. Christiernin, C. P. Clark, E. A. Colton, H. W. Cook, D. B. Cragin, G. E. Crawford, R. M. Daley, E. G. Dewis, E. J. Dewees, H. K. Dillard, H. W. Dingman, E. W. Dwight, O. M. Eakins, W. G. Extton, J. W. Fisher, H. M. Finnerud, Paul Fitzgerald, S. W. Gadd, F. I. Ganot, Arthur Geiringer, R. J. Graves, A. H. Griswold, F. L. Grosvenor, F. A. Hagney, G. C. Hall, J. B.

Thirty-Fifth Annual Meeting

Hall, Frank Harnden, Whitfield Harral, A. B. Hobbs, J. F. Honsberger, Arthur Hunter, Ross Huston, W. G. Hutchinson, Lefferts Hutton, C. B. Irwin, W. A. Jaquith, F. L. B. Jenney, A. E. Johann, G. E. Kanouse, J. E. Kinney, R. J. Kissock, A. S. Knight, E. B. Kyle, M. M. Lairy, W. P. Lamb, J. L. Larway, L. F. MacKenzie, F. H. McCrudden, C. B. McCulloch, T. F. McMahon, O. F. Maxon, Paul Mazzuri, S. W. Means, J. C. Medd, W. F. Milroy, J. T. Montgomery, J. H. North, Herbert Old, N. I. Olsen, J. A. Patton, W. O. Pauli, W. A. Peterson, J. S. Phelps, C. B. Piper, J. E. Pollard, W. E. Porter, J. T. Priestley, F. P. Righter, E. E. Rhodes, T. H. Rockwell, O. H. Rogers, F. W. Rolph, E. K. Root, R. L. Rowley, E. F. Russell, H. C. Scadding, C. E. Schilling, S. B. Scholz, Jr., D. M. Shewbrooks, J. M. Smith, T. A. Smith, Morton Snow, H. F. Starr, S. C. Stanton, J. B. Steele, Carl Stutsman, Lawrence G. Sykes, W. E. Thornton, P. E. Tiemann, Harry Toulmin, F. L. Truitt, J. P. Turner, Euen Van Kleeck, G. A. Van Wagenen, C. E. Waits, W. R. Ward, W. H. E. Weiner, F. S. Weisse, F. L. Wells, C. D. Wheeler, C. M. Whicher, C. F. S. Whitney, R. L. Willis, Gordon Wilson, M. C. Wilson, Glenn Wood, H. H. Young.

There were present also Drs. T. Stuart Hart, James Alexander Miller, F. B. Kingsbury, S. R. Benedict, E. L. Fisk, C. E. Skinner, A. R. Rose, Louis Dublin, G. P. Schadt, O. H. Folin, H. S. Wolf and Mr. J. D. Craig.

Total attendance at all sessions, 138.

On motion the roll call was waived and the members were requested to register their names in the book provided for that purpose.

On motion the reading of the minutes of the thirty-fourth annual meeting of the Association, held on October 18th and 19th, 1923, was waived.

The names of the following candidates recommended by the Executive Council for membership in the Association were presented:

Election of New Members

3

Dr. Walter C. Bailey, Assistant Medical Director, New England Mutual Life Insurance Company, Boston, Mass.

Dr. Henry A. Bancel, Medical Inspector, Mutual Life Insurance Company of New York, New York, N. Y.

Dr. Roland A. Behrman, Assistant Medical Director, John Hancock Mutual Life Insurance Company, Boston, Mass.

Dr. Wilton P. Blackford, Medical Director, Commonwealth Life Insurance Company, Louisville, Ky.

Dr. Frederick A. Causey, Associate Medical Director, Peoria Life Insurance Company, Peoria, Illinois.

Dr. Donald B. Cragin, Associate Medical Director, Aetna Life Insurance Company, Hartford, Conn.

Dr. Joe E. Daniel, Medical Director, Great Southern Life Insurance Company, Houston, Texas.

Dr. Ernest J. Dewees, Assistant Medical Director, Provident Mutual Life Insurance Company, Philadelphia, Pa.

Dr. Harold W. Dingman, Medical Director, The Continental Assurance Company, Chicago, Illinois.

Dr. Harold M. Frost, Assistant Medical Director, New England Mutual Life Insurance Company, Boston, Mass.

Dr. Henry W. Gibbons, Medical Director, Western States Life Insurance Company, San Francisco, Calif.

Dr. Robert J. Graves, Medical Director, United Life and Accident Company, Concord, N. H.

Dr. F. W. Hagney, Assistant Medical Director, Mutual Benefit Life Insurance Company, Newark, N. J.

Dr. R. M. Hargrove, Associate Medical Director, Great Southern Life Insurance Company, Houston, Texas.

Dr. Frank Harnden, Medical Director, Midland Mutual Life Insurance Company, Columbus, Ohio.

Dr. Daniel W. Hoare, Assistant Medical Director, The Penn Mutual Life Insurance Company, Philadelphia, Pa.

Dr. Byram Hollings, Assistant Medical Director, John Hancock Mutual Life Insurance Company, Boston, Mass.

Thirty-Fifth Annual Meeting

Dr. Francis H. McCrudden, Assistant Medical Director,
New England Mutual Life Insurance Company, Boston,
Mass.

Dr. C. E. Schilling, Medical Director, The Ohio State Life
Insurance Company, Columbus, Ohio.

Dr. Albert Seaton, Medical Director, American Central
Life Insurance Company, Indianapolis, Ind.

Dr. George H. Shaw, Assistant Medical Director, The
Travelers Insurance Company, Hartford, Conn.

Dr. Thayer A. Smith, Assistant Medical Director, Mutual
Benefit Life Insurance Company, Newark, N. J.

Dr. William B. Smith, Assistant Medical Director, Connecti-
cut General Life Insurance Company, Hartford,
Conn.

Dr. Samuel C. Stanton, Medical Director, Farmers Na-
tional Life Insurance Company, Chicago, Illinois.

Dr. Charles M. Whicher, Medical Director, Royal Union
Mutual Life Insurance Company, Des Moines, Iowa.

Dr. G. Elliott Woodford, Assistant Medical Director, Connecti-
cut General Life Insurance Company, Hartford,
Conn.

On motion, duly seconded and carried, the Secretary was
instructed to cast a ballot in favor of the election of each of
these candidates.

The Secretary announced the ballot so cast, and the candi-
dates were declared elected to membership in the Associa-
tion. The President appointed Drs. Whitney and Dwight
a Committee to introduce the newly elected members to the
Association. Eight of these members were present and they
were escorted into the room by the Committee, as follows:
Drs. Bailey, McCrudden, Cragin, Harnden, Shaw, Stanton,
Dingman and Whicher.

The Secretary announced that the following named gentle-
men were delegates from the Associate Companies:

Mr. Hardin's Address

5

Dr. M. M. Lairy, Lafayette Life Insurance Company, Lafayette, Ind.

Dr. H. M. Finnerud, Dakota Life Insurance Company, Watertown, S. D.

Dr. Carl Stutsman, Merchants' Life Insurance Company, Des Moines, Ia.

Dr. George E. Crawford, Cedar Rapids Life Insurance Company, Cedar Rapids, Iowa.

Dr. J. E. Kinney, Farmers Life Insurance Company, Denver, Colorado.

Dr. E. B. Kyle, Home Life Insurance Company, Philadelphia, Pa.

Dr. B. C. Brooke, Montana Life Insurance Company, Helena, Mont.

Dr. B. F. Byrd, National Life & Accident Company, Nashville, Tenn.

Dr. H. H. Young, George Washington Life Insurance Company, Charleston, W. Va.

The Secretary read the minutes of the meetings of the Executive Council of May 21st, 1924, and October 22nd, 1924. On motion these minutes were adopted as read.

Dr. Ward—I take pleasure, gentlemen, in introducing to you at this point in the proceedings Mr. John R. Hardin, President of the Mutual Benefit Life Insurance Company, a man who is in thorough sympathy with our work, and who will extend to you the greetings of our home office.

Mr. Hardin—Mr. President and Gentlemen: It is my very pleasant privilege to welcome you to the home office of the Mutual Benefit. It is perhaps appropriate, not only because for the time being your President is our own Dr. Ward, but I am told that our Dr. Van Wagenen is the sole surviving charter member of your Association.

The importance of Life Insurance in the business life of our country has become very great, and the relationship of the medical profession to that importance is very well un-

Thirty-Fifth Annual Meeting

derstood, nowadays. I was quite surprised to learn not very long ago, in reviewing some reports made to the Directors of this Company many years ago, that in 1877, when the late Judge Dodd, then Mathematician of this Company, and afterwards its President, visited England for the purposes of instruction, at the instance of the Company, he reported to the Directors that in England, although life insurance had very long been established, the medical profession was not considered of very much importance in conducting the business. The Actuary of the London Insurance Corporation, which had then been doing business for one hundred and thirty-six years, told Judge Dodd, that, in his opinion, the value of risks selected upon medical examination was very much over-rated, that such examinations could not, of course, be dispensed with, and risks taken in masses or groups, but if so taken, that their results would not differ much from those taken in the customary way, and, pointing to the stream of men upon the sidewalk, he said that it would be about as well to take them all in as to take the mass of lives selected under medical supervision.

Now, of course, he was mistaken as to the importance of the medical profession as a part of the machinery in the selection of risks, and in these days the importance of the medical side has been abundantly indicated. There are at work throughout the life insurance companies now many co-operative agencies for the general good of the business and none are more important than this society of yours. Being men of education and high attainment, you are able to co-operate with each other on a plane of association which is sure to secure the best results. It would be very inappropriate for me to detain you from your important work, but I trust that your stay with us may be pleasant, that your conferences may be agreeable and advantageous, and that your work may further cement the very friendly relations between yourself and the companies you represent.

Memorial of Dr. Burr

7

Dr. Ward—Gentlemen, as we meet today we are reminded of the fact that there are those who have met with us for many years, who themselves are not here this morning. But yesterday they were with us, giving us their counsel and their fellowship. Now they have gone hence, but they have left to us a priceless legacy because of the splendid work that they have performed. They have left also the memory of a friendship which will endure forever. During the past year, the following members of our Association have died:

Dr. Chauncey R. Burr, of the Metropolitan Life Insurance Company.

Dr. Z. Taylor Emery, of the Manhattan Life Insurance Company.

Dr. Ignatius Haines, of the John Hancock Mutual Life Insurance Company.

Dr. William W. Knight, of the Connecticut General Life Insurance Co.

Dr. Brandreth Symonds, of the Mutual Life Insurance Company of N. Y.

Dr. Franklin C. Wells, of the Equitable Life Assurance Society.

Memorials have been prepared on all of these departed members and will now be presented to the Association.

The following memorials were read and on motion ordered to be spread upon the minutes of the Association. After the reading of the memorials the members stood for a moment in silent tribute to the men who have passed away.

DR. CHAUNCEY RAE BURR.

1862-1923.

(Presented by Dr. Christiernin.)

Doctor Chauncey Burr entered the service of the Metropolitan Life Insurance Company as Assistant Medical Director in 1917 and in the following year became a member of

Thirty-Fifth Annual Meeting

this Society. Doctor Burr took his bachelor's degree at Yale in 1884 and his degree in medicine at Harvard in 1888. He continued his studies at Heidelberg, at St. Barthelme's Hospital in London and followed this with an internship at the Rotunda Hospital in Dublin.

Upon returning to the United States he took up practice and became Assistant Physician in the Department of Diseases of the Nervous System, at the Boston City Hospital. In May, 1898 he volunteered and was commissioned as Assistant Surgeon in the United States Navy for temporary duty, and saw service in the Battle of Manila Bay. He continued in the Navy until September, 1901, when he resigned and resumed practice in Portland, Maine, where he remained until his entry into Insurance Medicine with the Metropolitan Life Insurance Company. His efforts were largely given to Disability Insurance a subject which was of interest to him, having published in the records of the 63rd congress a treatise on, "The Economic Value of Man."

Doctor Burr was born in Portland, Maine, and came from old New England stock. He was of a quiet, retiring nature, gentle and considerate in his relations to others. His death on December 24th, 1923, came as a great shock to his many friends of the Metropolitan Life Insurance Company where he was held in high esteem by all who knew him.

DR. IGNATIUS HAINES.

1871-1924.

(Presented by Dr. Allen.)

Dr. Ignatius Haines, Medical Director in Chief of the John Hancock Mutual Life Insurance Company died at his home in Brookline on January 16, 1924. His last illness was of short duration and his death was sudden and unexpected.

Memorials of Dr. Haines and Dr. Emery 9

Dr. Haines was born in Cambridge, Massachusetts, Nov. 15, 1871. He received his early education in the Cambridge schools and graduated from the Harvard Medical School in 1897. He practiced medicine in Cambridge until 1903 when he gave up active practice to make insurance medicine his life work.

Dr. Haines was appointed an examiner for the John Hancock Mutual Life Insurance Company in April, 1901, and joined the Home Office staff in July, 1903. His early years with the Company, first as Weekly Premium Examiner and later as Medical Inspector in the field gave him a deep insight into the business of life insurance. For many years he traveled extensively and built up a large acquaintance both with the field examiners and with the agency staff. He was appointed Associate Medical Director in 1920 and Medical Director in Chief in January, 1923.

Dr. Haines was always kind, courteous and considerate of those about him. He was an able organizer and a natural leader with broad vision and sound judgment. He had a fondness for the theory rather than for the practice of medicine and was able to turn his knowledge into the paths of preventive medicine and hygiene. He was in addition a delightful companion and a firm and loyal friend. His sudden death at the height of his mental and physical vigor did not permit him to round out a full life of service and usefulness.

DR. Z. TAYLOR EMERY.

1847-1924.

(Prepared by Dr. Barber.)

Dr. Z. Taylor Emery, Medical Director of the Manhattan Life Insurance Company for a period of twenty-eight years, died at his late residence in White Plains, New York, October

Thirty-Fifth Annual Meeting

6th, 1924, following a cerebral hemorrhage due to a continual hypertension, which he had been constantly fighting for several years.

Dr. Emery was born in Lyon township, Oakland County, Michigan, February, 1847. He was educated in the local schools of the county where he later taught, until he became a student at the University of Michigan, both in the academic and medical departments, but later received his M. D. degree from the Detroit College of Medicine, now the Detroit Medical College, in 1874, and the following year from the Long Island College Hospital, Brooklyn, New York. He practiced his profession in Brooklyn 1874-1905, and was appointed Commissioner of Health of Brooklyn, 1894, and served in this capacity until 1907. He became Medical Director of the Manhattan Life Insurance Company 1895 and served until a year before his death. He was president of the Kings County Medical Society 1891-92, member Associated Physicians of Long Island and the American Medical Association and The Practitioners Club of Brooklyn.

As an active and successful practitioner of medicine in Brooklyn for thirty years, Dr. Emery represented that type of general practitioner; a friend of his patients; loved and respected by his profession; the kind so lacking yet so needed in the more populated communities of today. It was he who as Health Commissioner compelled the vaccination of about 500,000 Brooklynites and so terminated a smallpox scare; it was he who enlarged and improved conditions in the Kingston Avenue Hospital for contagious disease in Brooklyn and started the movement toward the development of it as a modern and great hospital, which it is today. He called upon the citizens of Brooklyn to give aid, which Aldermen had refused, to lessen infant mortality, received it and used it efficiently. At the end of his term as Health Commissioner, Brooklyn had the lowest death rate in its history. As attending physician to the Kings County Hospital of Brooklyn, Dr.

Emery rendered a service for charity; at the same time his energies as a member of the medical board of that hospital were of material help in the growth and development of the hospital, making that institution one of our most modern in equipment and one of our great hospitals of today. Retiring from active service he became one of its consulting physicians on the Medical Service and served in this capacity until his death. These years of service as a general practitioner peculiarly fitted him for his work as medical director and his rich experience and rare judgment utilized in examination and selection of risks were respected by all of his associates.

A representative of the old school, yet well astride of his contemporaries, Dr. Emery will ever be remembered for his genial personality and sterling character.

DR. WILLIAM WARD KNIGHT.

1852-1923.

(Presented by Dr. Sykes.)

Dr. William Ward Knight, a Medical Adviser of the Connecticut General Life Insurance Company from May, 1899, until his death, died December 4, 1923, in the seventy-second year of his age, after a faithful and valued service of nearly twenty-five years.

Born in Chaplin, Connecticut, September 15, 1852, he graduated from Bellevue Medical School, New York University, in 1876, and for about twenty years practiced medicine in Hartford. At one time he was on the consulting staff of the Hartford Hospital and also President of the Hartford Medical Society.

On May 2, 1899, he became connected with the Connecticut General as Assistant Medical Adviser and a year later, upon

Thirty-Fifth Annual Meeting

the death of Dr. Melanthon Storrs, he became Medical Adviser.

A member of this Association since 1901, some of you present will remember him as modest and retiring in disposition, known intimately by a comparatively small number, but those who did share his friendship knew him as a man of sound judgment, broad sympathies, absolute fairness and unfaltering courage.

Although he was a French scholar and interested in art and architecture, he was chiefly devoted to the science of medicine, as illustrated in a bequest of \$10,000 to the Hartford Medical Society, the income from which is to be used "in obtaining the services of eminent physicians, surgeons and men otherwise learned in the science of medicine, to address said Society from time to time."

During his long connection with the Connecticut General Life Insurance Company he exhibited a high degree of skill in the examination of and the selection of risks and was highly regarded by his associates.

His death was a matter of keen regret to his friends and associates, and to the Company in the loss of a competent and trusted adviser.

DR. BRANDRETH SYMONDS.

1863-1924.

(Prepared by Dr. Porter.)

During the past year this Association has lost one of its most valued members. Dr. Brandreth Symonds, who was elected to membership at the 10th Annual Meeting, held at the offices of the Mutual Life Insurance Company of New York, May 31st, 1899. Dr. Symonds was born in Sing Sing, New York, March 4th, 1863, the son of Col. Henry and Beatrice Brandreth Symonds. He had his collegiate education

at Hobart College, receiving the degree of A. B. in 1881, A. M. in 1884 and LL.D. in 1921. He attended the College of Physicians and Surgeons, New York, graduating in the Class of 1884; served on the house staff of Bellevue Hospital during 1885 and 1886; later he held the following positions: attending physician, Bellevue Hospital Dispensary, Diseases of the Heart and Lungs, 1886-1887; Northwestern Dispensary, 1886-1888; Instructor at the New York Polyclinic, General Medicine and Diseases of the Chest, 1887; Lecturer on Life Insurance Medicine at the University and Bellevue Medical College; and the College of Physicians and Surgeons, 1907-11; Assistant Attending Physician, Roosevelt Hospital Dispensary, 1888-90. He was appointed Medical Examiner for the Mutual Life Insurance Company of New York in 1889, becoming Chief Medical Director in 1907. He served as Secretary, Vice President and President of this Association, contributing liberally to the scientific work throughout the entire period of his membership. Among his numerous contributions may be mentioned the following: Undergraduate Instruction in Making Life Insurance Examinations; A Statistical Study of Renal Colic and Hepatic Colic; Some Studies in Family History; Report of Committee on Bubonic Plague; Statistical Report on the Influenza Epidemic as it Affected the Policy Holders of the Mutual Life Insurance Company of New York in the Year 1918 Only; Value of the Medical Examiner's Opinion; Blood Pressure of Healthy Men and Women. He was author of "A Manual of Chemistry for the Use of Medical Students"—1889, and "Life Insurance Examinations"—1905. He was an active Episcopalian, a Member of the Loyal Legion, Colonial Order of the Acorn and Sigma Phi Fraternity; a Fellow of the New York Academy of Medicine, a Member of the American Medical Association, the New York State and New York County Medical Societies and the Society of the Alumni of Bellevue Hospital. He was a Member of the Grolier and Church Clubs.

Thirty-Fifth Annual Meeting

The excellence of Dr. Symonds' work as a member of the various Committees on which he served, especially the Medico-Actuarial and M. I. B. Committees, is well known to all and he was always a valued and liberal contributor to the discussions of this Association. He had been in poor health for some years and died on August 10th, 1924, at his residence, #33 Central Avenue, St. George, Staten Island, New York. He is survived by his widow, Florence Bacon Symonds, and also by a son, Brandreth Symonds, Jr.

DR. FRANKLIN C. WELLS.

1860-1923.

(Presented by Dr. Rockwell.)

"He had kept the whiteness of his soul,
and thus men o'er him wept."

—Lord Byron.

Franklin C. Wells, of sterling ancestry, was born in Chicago, where his boyhood was spent and where the example of his home life greatly influenced his career. Finishing his college life he went to New York, began the study of medicine at Bellevue, graduating from there in 1887. He chose Charity Hospital for his internship and finished his service in September, 1888. It was there that I first met him, and then began an association both intimate and affectionate which lasted more than thirty years.

As a young man he was very undecided as to whether he should devote himself to ecclesiastical work or study medicine, but his choice of the latter in no wise lessened his interest in church and religion. Always a devout man, and deeply concerned not only in his own future life, but also in that of others, it was natural and opportune that he and his young bride, Clara Downs, should embrace the opportunity of associating themselves with the religious and medical work of Roberts College, Beirut, Syria. Here they labored for nearly

three years, then returned to Chicago, where Dr. Wells began his active practice in 1891.

Soon after he became an examiner for the Equitable Life Assurance Society.

In 1903 he was induced to enter the Home Office at the head of the Inspection Department. In 1905 he was made Assistant Medical Director, and in 1909 Medical Director.

In 1916 he was placed at the head of the Health Conservation Department, in which position he continued until his death. He also had charge of the medical work of the Group Insurance Department and in both of these he was an unqualified success. He issued many bulletins and timely warnings to the policyholders to induce them by right living, proper hygiene, and self control to correct the error of their ways and aim to attain longevity.

To the Equitable Group patrons he was especially devoted, and as his eloquence as an orator was well known, he was constantly in demand to address workmen in factories, machine shops railroad terminals, as well as banks, financial corporations and officers in charge of the employment of vast numbers of men and women.

His versatility and ease of manner were such that he appealed to men in all sorts of positions of life, and he was equally at home in addressing a crowd of laborers or a board of directors. And when he finished his talk he was always enthusiastically applauded and usually requested to come again.

Members of this Association remember with pleasure his after dinner speeches, and the grace with which he presided during his term of office in 1915-1916.

His home life was beautiful and ideal. Of a benevolent and generous disposition, full of great family love, ever looking out for the interests and happiness of his friends, both in business and in his church, he was highly venerated by those who knew him.

Thirty-Fifth Annual Meeting

DR. WILLIAM R. WARD, President of the Association, then delivered the following address:

Fellow Members: At the annual meeting it is customary for the President to address the members of the Association. This address is usually a presentation of a scientific subject or a report of the study of some impairment or group of impairments that has been made by the President or by the Company that he represents. As I have approached this task, I have pictured this group of new men who have come into our Association, and it has seemed to me that it might be of advantage to us at this time if we would depart somewhat from our usual custom, and if I were to speak to you for a short time this morning concerning the record of our Association, endeavoring to emphasize some of the things that we have accomplished, and then, if possible, to point out some new fields that lie before us.

It was thirty-five years ago, in 1889, that Dr. John M. Keating, Medical Director of the Penn Mutual Life Insurance Company, recognized the advisability of establishing an Association of this kind. Consequently he invited a group of Medical Directors to confer with him, and on the 29th of May of that year (1889) this group of twenty-eight men representing twenty-one Companies met in the Union League Club of New York City. They realized the advantages that were to accrue from such an organization and consequently they appointed a Committee to draft a constitution and to report at a later meeting. In December of that year, this Committee was ready to present a Constitution and to submit its report. As has been said by Mr. Hardin this morning, there is only one surviving member of that group, and later, at one of our sessions, we are going to be favored with some reminiscences by Dr. Van Wagenen, as to the men who organized our Association. Suffice it for me to say that they were men of outstanding qualifications; men who stood high in their profession. Dr. Keating was elected the first Presi-

dent. Dr. Keating was not only a physician and a noted pediatrician, but he was an author as well. Many of you may have read his Encyclopedia of the Diseases of Children. As we think of that group, we realize the benefits that have come to us because of the type and character of those men.

Then, too, it is of interest to us at this time to realize the objects of this organization, as set forth in that original constitution. Three objects were stated: First, the promotion of medical science as applied to life insurance; second, the encouragement of social and friendly relations between the members; and, third, the advancement of the general interests of life insurance.

Before we proceed further, let us compare life insurance as it was in 1889, and life insurance as it is today. The figures are so amazing that they fairly stagger us. In 1889, there was in force in the life insurance companies of the United States and Canada, not including the fraternal or assessment companies, less than four billions of dollars (\$3,889,391,351.00). At the close of 1923 there was in force in the United States and Canada, not including the fraternal or assessment companies, more than seventy billions of dollars (\$70,344,380,816.00), an increase of over 1700%. Then, too, contrast the amount of business written at that time and compare it with the amount written today. In 1889, the year in which our organization came into existence, the amount written by the life companies of the United States and Canada was approximately one billion dollars (\$1,064,021,783.00). During last year the amount was over fourteen billion dollars (\$14,148,234,208.00), an increase of over 1300%.

These figures fairly stagger the imagination. Not only do they show the extent to which life insurance has spread, but they also show the recognition of this universal need and the acceptance of this need by the people. They impose upon us a responsibility which it is difficult for us to measure.

18 Thirty-Fifth Annual Meeting

Such was the beginning of our organization. And now let us inventory some of its accomplishments.

In the first place, year by year, these men and their successors have brought to us studies of impairments, studies prepared by their companies at a great deal of expense and a great deal of labor, and in the ten volumes of our transactions they have presented to us for our guidance reports upon almost every medical subject that comes within the range of life insurance. The value of this work it is difficult for us to estimate. To this task these workers, many of whom have long since departed from us, have given the best that they possessed. By their indefatigable toil they have left to us a rich legacy for our instruction and guidance. While I recognize the value of all of this service, I wish to speak of three things in particular that have been accomplished by the Members of our Association. The first thing that I would refer to is the work of the Medico-Actuarial Committee. There came a time when our organization realized that better work could be done by group co-operation than by individual companies working alone. Let me preface the statement concerning the work of the Medico-Actuarial Committee by telling you of the work done by the Actuaries prior to that time.

In 1901 the Actuarial Society realized the necessity for a mortality study, and so they appointed a Committee and made such a study. The material for that study was supplied by thirty-four companies, extending over a period of thirty years, from 1870 to 1900. The report made by these Actuaries was called "The Specialized Mortality Report." It included ninety-eight subjects and it gave to us most valuable data for our guidance in the selection of risks. As time went on, both the Actuaries and the Medical Directors realized that this data, valuable as it was, could be supplemented and made still more useful by a report made neither by the Actuaries alone nor by the Medical Directors alone, but by a Joint

Committee, and so, on the 15th of June, 1909, a Committee met in New York City, consisting of a group of Actuaries and three members of our organization, Dr. Dwight, Dr. Rogers, and Dr. Willard. A fortnight later a second meeting was held at which time our Association was represented by Dr. Rogers, Dr. Dwight and Dr. Toulmin. It is impossible for those who have not been intimately in touch with that Committee to realize the vastness of its work. Let me give you two or three facts concerning it:

In the first place, it embraced the study of two million policies supplied by forty-three Companies, including all of the leading Life Insurance Companies in the United States and Canada, and covering a period of twenty-four years. When the five volumes that comprised the report of this Committee were presented to us, we realized that a great step had been taken in life insurance, for it was the most exhaustive and the most comprehensive report that had ever been presented. Not only has that report been of great benefit to life insurance, but it has been of benefit to medical science, and to humanity in general, for it has shown to the world the deleterious effect of excessive weight, of unfavorable occupation, of the excessive use of alcohol, and of other conditions concerning which we formerly had very indefinite knowledge, but concerning which we now have a knowledge based upon the study of this great number of lives.

The next service that I would refer to are the studies in blood pressure, for this Association has done a great deal in that field. It was as far back as 1905, at the sixteenth Annual Meeting, that Dr. Root referred to the value of blood pressure in differentiating organic from functional heart murmurs. At the next meeting of our Association, Dr. Root described to the Association the various instruments for taking blood pressure which were then in use. Rather crude they were at that time, compared with the perfected instrument of today; and at that meeting, in 1906, Dr. Rogers made

the very significant remark that the time was not far distant when blood pressure would be of greater value to medical examinations for life insurance than the analysis of the urine. Five years later, Dr. Fisher gave to us that very splendid report which marked the real beginning of our blood pressure study. In that report he gave to us the results of a study of 13,000 cases that had been examined for the Northwestern Mutual Life Insurance Company from the year 1907 to the year 1911, and accepted as standard risks. By the study of those 13,000 risks, Dr. Fisher was able to give to this Association, and through this Association, to the profession and to the world, the average systolic pressure. More than that, Dr. Fisher at that time had secured sufficient mortality data to show the injurious effect of high blood pressure and to recommend what he considered a normal range. From that meeting to the present time, this subject has had a very prominent place in every program, and you gentlemen are fully aware of the contribution that we have made to medical science because of this work.

One other accomplishment—one that I think is very great—I desire to present to you. Prior to 1922 this Association consisted only of what we might call the larger Companies, those whose business for five years prior to their admission had been not less than five million dollars annually. But some of our members—Dr. Rogers and Dr. Knight were prominent in that group—realized the great value that would accrue to life insurance in general if the smaller Companies, which were of good repute, but were not writing business to that extent, might be included as Associate Members; and so by their efforts and by the co-operation of these other Companies, we formed an Associate Membership Group, and the delegates from that group are with us today. The basic requirements for admission to that group are that the Company must be of good repute, must conduct its business upon the level premium legal reserve plan, must have a Medical Direc-

tor in charge of its medical affairs, who is responsible for the confidential nature of the correspondence.

These are only a few of the things that our Association has accomplished.

The second object in organizing this Association was the promotion of a cordial relationship between the members. Your commingling today is an evidence of the fulfillment of that desire. Prior to that time our acquaintance with one another was very limited and consequently our correspondence was most formal, but today we know how glad we are to help one another in our daily tasks, how we give to one another information that we possess, not grudgingly, but willingly, for all of us are only too anxious to supply it.

And now, my fellow members, what of the future? That is the great question that confronts us at this time. Let us realize that we are not only Medical Directors, but all of us are physicians as well—men who have dedicated their lives to the art of healing. As physicians, sometimes we are discouraged because of the slowness of our progress. We look upon the great problem of cancer, a problem that our investigators and research men have been studying year after year, and yet, notwithstanding this exhaustive study, it is a fact that at the present time approximately one-tenth of all the deaths after forty years of age, are due to cancer. We look upon pneumonia, and somehow we feel that we are upon the threshold of a better day in the conquest of that disease, and yet we know the appalling mortality that we still experience because of pneumonia. When the Influenza Epidemic spread over our land in 1918, we felt our weakness, even our utter helplessness, for the very flower of our manhood and womanhood was stricken down before our eyes.

These things have confronted us and at times have tended to dishearten us, but let us realize that this is only one side of the picture, that the credit sheet is much larger than the debit, for during these past thirty-six years we have seen some

marvelous accomplishments. For example, let us think of Typhoid. Thirty-five years ago the mortality from Typhoid in our Northern Cities was 47 per hundred thousand. The last census report shows this to have been reduced to 6.8 per hundred thousand. You know that the occurrence of Typhoid Fever at the present time is an indictment against the efficiency of the health service of our cities. Then think of the great white plague, Tuberculosis. Thirty-five years ago the mortality from Tuberculosis was 247 per hundred thousand. The last report which I have seen—the 1923 census report—showed that the mortality had been reduced from 247 to 93.6 per hundred thousand. It is well for us to realize that this means a saving of one hundred and fifty thousand lives annually in our country. Then let us think of Diphtheria. In 1889 the mortality from that disease was 135 per hundred thousand. By the discovery and use of antitoxin, this mortality has been reduced to a mortality of 12 per hundred thousand. You men know the benefit we are receiving from the use of the Schick test in the public schools, a benefit so marked that we may confidently predict that the mortality of 12 per hundred thousand will in time be cut down to half that figure.

Then think of that other great scourge—Yellow Fever. For three hundred years it was the scourge of the Tropics. Not only was it the scourge of the Tropics, but at frequent intervals it was carried across the Gulf and it then invaded our Southern cities. During these various epidemics, not less than forty thousand of our American citizens died of Yellow Fever. Now it is only a memory, for it is a disease which has been absolutely conquered.

But these achievements, marvelous as they are, are not more marvelous than is the reduction of infant mortality. Thirty-five years ago, during the first year of life, the mortality was three hundred per thousand. One-third of all children died during the first year. This has now been reduced to

less than half that figure. All these advances have reduced our death rate from a death rate of 19 at that time to a death rate of 12.3 at the present time. These results have been accomplished by many factors, but the indefatigable toilers in our Laboratories and the Clinicians at our bedsides have contributed a large measure of this result. Such figures as these should give us, as Physicians, a great deal of encouragement as should stimulate us to renewed efforts.

And now I am thinking of our work as Medical Directors for the coming year. What shall it be? I believe that we should tread the same paths that we have been treading. We shall continue to investigate disease and its effect on mortality, and then we will give to all the Companies of our Association the data which we as individual Companies may have compiled.

Then, I have another thought for you men. To my mind a very important thought, and one that I want to impress upon you more than all else. A short time ago, Mr. Job Hedges, the Counsel for the Association of Life Insurance Presidents, had occasion to speak to the National Underwriters Association at Los Angeles. Mr. Job Hedges is a corporation lawyer, many of you may know him. When he went before that Association, the central thought of his address was not to tell them of the difficulties he was having because of different legislation in forty-eight different States, nor was it to explain to those men the legal aspects of the life insurance contract, but this was the message which he gave, and I want you to take it with you this morning as the keynote of this Convention:

"I say very seriously, as the result of deep contemplation, that I do not believe that the brain is the generating factor of the human race. All the big missions that have been accomplished in life, the Crusades, the great discoveries, have originated in the heart and been regulated by the brain."

These words are not from a visionary sentimentalist, nor

Thirty-Fifth Annual Meeting

from a Utopian dreamer; they are from the man who has been selected by the Presidents of our Companies to represent their Association as their Counsel. It was his desire to impress upon that Association of Life Insurance Underwriters the importance of the human element in our lives. I feel that we should introduce into our work more of this human element.

But how may it find expression? Let me suggest one or two opportunities along this line. You have met today in the Home Office of the Mutual Benefit. We have in this building approximately five hundred employees, men and women. You men represent Companies, many of which have much smaller groups. Some of you represent Companies with groups ten times as large. These people are our closest neighbors. They need our protection. It is our function and privilege as Medical Directors to assist our Executives in seeing that the rooms that they occupy are not overcrowded, if possible to see that they are supplied with a suitable noonday meal, and when they are sick to see that they are not neglected. We should also endeavor to so safeguard their future that when they are taken from us those whom they leave behind, and who have been dependent upon them shall receive suitable compensation. That thought I leave with you. It must find its expression in your various Companies according to your ability and your needs, but I want to lay that burden upon you.

Then I am thinking of that still larger group—Oh, so much larger—the policyholders of our companies. Let us not think of them as mere payers of premiums, people who came to us sometime ago and who continue on our books until they pass off as death claims, but let us realize that we, as Medical Directors, have a splendid opportunity of rendering a service to them. I am not proposing at this time any ways or means. Some plans may come before you during the sessions of this Convention. I simply want to lay this

Report of Nominating Committee 25

burden also upon you, that you may realize the opportunity that is yours, and I hope that you will not neglect this great service.

And now in closing, I want to say to you that I appreciate more than I can express the honor that you have conferred upon me in electing me as your President. As I retire from this office, the one thought I want to leave with you more than all else is that you may take home with you this human element in our work, that you may see the splendid opportunity that is yours, that you may keep in close touch with the friends in your Home Office, and with the great group of policyholders throughout the field at large; in short that you may "live in a house by the side of the road and be a friend to man."

The Nominating Committee presented the following report:

"The Nominating Committee begs leave to make the following report:

"At a meeting held October 22, 1924, the Committee by votes duly seconded and carried decided to recommend the nominations of the following:

For President—Dr. Chester F. S. Whitney,

For First Vice President—Dr. Angier B. Hobbs,

For Second Vice President—Dr. Wesley W. Beckett,

For Secretary—Dr. Chester T. Brown,

For Treasurer—Dr. Charles L. Christiernin,

For Editor of the Proceedings—Dr. Eugene F. Russell,

For members of the Executive Council: Dr. G. A. Van Wagener, Dr. E. W. Dwight, Dr. J. Allen Patton, Dr. William Muhlberg, Dr. Ross Huston.

All of which is respectfully submitted,

(Signed) W. M. EVELYN PORTER,
C. F. S. WHITNEY,
R. M. DALEY,
HARRY TOULMIN,
CHESTER T. BROWN,
THOMAS H. WILLARD, Chairman."

Thirty-Fifth Annual Meeting

Dr. Ward—According to our by-laws the election of these officers will take place tomorrow morning. It is in order at this time to receive from the members any additional nominations. If you have no names to present, it will be in order to move that the nominations be closed.

No further nominations being presented, it was moved by Dr. Rockwell and seconded by Dr. Jaquith that the nominations be closed, and that the Secretary be instructed to cast a ballot for the officers and members of the Executive Council placed in nomination by the Nominating Committee. The motion was carried.

The Treasurer, Dr. C. L. Christiernin, read his report. The Auditing Committee, Dr. Whitney and Dr. Jaquith, reported that the Committee had audited the Treasurer's Report and found it to be correct. On motion, duly seconded, the report of the Treasurer was accepted with thanks and placed on file.

Dr. O. H. Rogers, Chairman of the Special Committee in charge of the M. I. B. presented the report of that Committee, which was accepted with thanks, and ordered placed on file.

On behalf of Dr. Willard, Chairman of the Committee on Public Health, the Secretary reported that nothing had occurred during the past year to require the action of the Committee, but the Chairman of the Committee recommended that the Committee be continued, and that appointments be made to fill the vacancies on the Committee caused by the death of Dr. Symonds and the death of Dr. F. C. Wells.

On motion by Dr. Jenney, seconded by Dr. Jaquith, the report of the Committee on Public Health was accepted and placed on file.

The Committee appointed by the Executive Council to consider the advisability of assessing the Associate Membership Companies an annual fee, reported as follows:

"In view of the expense incident to the administration of

Report of Committee on Assessment 27

the affairs of the Medical Information Bureau, in connection with Associate membership, by the Association of Life Insurance Medical Directors, the following motion is recommended:

Moved that each Associate Member Company of the Medical Information Bureau be requested to pay an annual assessment of Five Dollars to the treasury of the Association of Life Insurance Medical Directors, to become effective on January 1, 1925.

(Signed) A. B. HOBBS,
C. T. BROWN,
C. L. CHRISTIERNIN, Chairman."

It was moved and seconded that the report be adopted. The President called for an expression of opinion from the representatives of the Associate Member Companies. The delegates who were present spoke on the motion, and expressed appreciation of the benefits derived by the Associate Member Companies from the Association, and were unanimous in the opinion that those Companies would be glad to meet such annual charge. The motion was carried.

Dr. Ward—Will Dr. Dublin present the Report of the Dreyer Committee?

Dr. Dublin—Mr. President and Gentlemen of the Association—I hope that you will allow me to spread on your minutes a word for our Committee expressing its deep sense of loss in the death of its Chairman, Dr. Symonds. I know perfectly well that you will do honor to the memory of Dr. Symonds, but this Committee had its origin in a motion made by our dear friend, who threw himself keenly into the work of the Committee, and expected to find things from it. I know that every member of the Committee has felt very deeply the loss of his advice and of his guidance this year. If it is proper for me as an individual who has contributed somewhat to the field of medical statistics, to speak personally, I may say that if I have contributed anything in these last twenty years, it is because of the inspiration and guidance of that great man. I learned everything from him. I will now present the report of the Committee.

**1924 Report of the Committee on Dreyer
Measurements in Relation to Life Insurance
Underwriting Practice**

The Committee has this year conducted an investigation into the possible use of spine length measurements in the assessment of physical fitness. The investigation was confined to overweights and, specifically, the Committee attempted to determine whether such overweights gave better or worse mortality in accordance with variations in the length of the spine.

Reference to Table 7 of the Committee's Report for 1923 will show that there were 38,062 overweights of all build classes in the material turned over to the Committee by the Union Central for study. Because of the small number of entrants in overweight build classes IV and V, the detailed mortality investigations were applied only to classes I, II and III. Even build class III did not show a sufficient and dependable exposure in some of the subordinate tables for spine length classes, and the calculations of expected and actual mortality were in such few instances omitted.

The cards in each overweight build class were further segregated for (a) short men, that is, heights 66 inches and less; (b) men of medium height, heights 67 to 70 inches, and (c) tall men, heights 71 inches and over. The following table shows the volume of our basic material.

TABLE I.
*Number of Overweight Men in Specified Build and Height Classes,
Union Central Policy Issues, 1887 to 1907.*

| Build Class | ALL MEN | | SHORT MEN | | MEN OF MEDIUM HEIGHT | | TALL MEN | |
|-------------------|---------|---------------------------------|-----------|---------------------------------|----------------------|---------------------------------|----------|---------------------------------|
| | Number | Per cent. of total over-weights | Number | Per cent. of total over-weights | Number | Per cent. of total over-weights | Number | Per cent. of total over-weights |
| All over-weights. | 38,062 | 100.0 | 6,567 | 100.0 | 22,888 | 100.0 | 8,607 | 100.0 |
| Build Class: | | | | | | | | |
| I..... | 25,932 | 68.1 | 4,390 | 66.8 | 15,866 | 69.3 | 5,676 | 65.9 |
| II..... | 9,494 | 24.9 | 1,670 | 25.4 | 5,560 | 24.3 | 2,264 | 26.3 |
| III..... | 2,323 | 6.1 | 450 | 6.9 | 1,279 | 5.6 | 594 | 6.9 |
| IV..... | 304 | .8 | 55 | .8 | 180 | .8 | 69 | .8 |
| V..... | 9 | — | 2 | — | 3 | — | 4 | — |

Report of Committee on Measurements 29

More than two-thirds of the overweights (25,932) were in build class I, where the amount of overweight was approximately 16 to 20 pounds above the norm assumed in the modified Shepherd Table.* Approximately one-quarter of these overweights (9,494) were in build class II, where there was an excess of some 32 pounds above the Shepherd norm. Build class III contained between 6 and 7 per cent. of the overweights (2,323). Classes IV and V contained negligible numbers for purposes of this study.

We next classified each height-build group according to relative spine length, *i. e.*, the percentage spine length of total height. (Calculations of relative spine length had been made by the Union Central office staff and were available on the punch-cards supplied to the Committee). While the anthropometric statistics shown in our 1923 Report for relative spine length were based upon narrow size classes in order to increase precision in the measurement of the constants of the distributions, we adopted for the mortality investigation three broad classes for the relative spine length observations. Having determined last year the standard deviation of each of the distributions of relative spine length for the overweight-height-build groups, we used the calculations of standard deviation for each series in setting up the following working procedure:

CLASSIFICATION OF RELATIVE SPINE LENGTHS FOR THE MORTALITY TABLES.

For overweight men we had three height classes, and for each of these height groups six build classes—the five definitely specified builds and the total for all builds combined. This made eighteen height-build groups. Now, for each of these basic divisions of the data we were to determine the extent to which the mortality varied according to relative spine length; that is to say, for overweight short men in build class I, what was the ratio of actual to expected mortality for men with short spines, with spines of medium or average length, or with long spines? The definitions of "short," "average or medium" and "long" spines were established as follows:

*Pages 120-121, Vol. 1, Medico-Actuarial Investigation.

For each of the eighteen basic tabular height-build units we knew (1) the mean relative spine length and (2) the standard deviation of these relative spine lengths. These values had been computed for the tables of anthropometric statistics presented in the 1923 report.* "Average or medium" relative spine lengths were said to be those not varying from the mean relative spine length by more, plus or minus, than the value of the standard deviation. Values which fell below the lower limit of the "average or medium" relative spine length group were placed in the "short spine" class, and those falling above the upper limit were considered to be in the "long spine" class. While this tri-part division was made for the cards of each of the eighteen basic tabular units above defined, the counts of cards showed that calculation of a mortality table for each build-spine-length class would not be feasible. Data were sufficiently abundant to warrant the preparation eventually of thirty-two tables, and the final results for each table—the number of actual deaths, the number of expected deaths according to the M. A. select and ultimate table, and the ratio of actual to expected mortality—are shown in result tables 2, 3, 4 and 5 of this report.

Throughout the compilations, the staff followed the tabulating procedure outlined for the Medico-Actuarial Investigation, and for others of like character, with the single exception that all issues were entered upon the basic tables at central points of five-year age groups and that the attained ages of the exposure were calculated by single year increments from these central age points. This procedure considerably abridged what was at best a tedious, laborious task. At every step, suitable checks were made to insure accuracy of the tabulations and of secondary calculations.

While ratios of actual to expected mortality were calculated for (a) aggregate (b) select and ultimate, (c) select and (d) ultimate exposure ranges, only the select and ultimate results are shown in the following text and tables:

**Proceedings, Thirty-fourth Annual Meeting of the Association of Life Insurance Medical Directors*, Table 12, page 179.

Report of Committee on Measurements 31

TABLE 2.
Comparison of Actual and Expected Mortality among Overweights, 1887-1908. By Height Class and Relative Spine Length. White Male Issues of Union Central Life Insurance Company.*

ALL BUILD CLASSES.

| BUILD CLASS AND RELATIVE SPINE LENGTH | ALL HEIGHT CLASSES | | OVERWEIGHT SHORT MEN | | OVERWEIGHT MEN OF MEDIUM HEIGHT | | OVERWEIGHT TALL MEN | |
|---|--------------------|-----------------|---------------------------|---------------|---------------------------------|---------------------------|---------------------|-----------------|
| | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths |
| All build classes!..... | 4,571 | 4,299.2 | 106.3 | 786 | 760.7 | 103.3 | 2,683 | 2,582.5 |
| Relative spine length: | | | | | | | | |
| Above limit of S. D. + from mean..... | 493 | 468.2 | 105.3 | 117 | 108.4 | 107.9 | 286 | 271.0 |
| Within limits of S. D.+ or — from mean..... | 3,573 | 3,334.3 | 107.2 | 596 | 575.7 | 103.5 | 2,107 | 1,998.2 |
| Below limit of S. D. — from mean..... | 505 | 496.7 | 101.7 | 73 | 76.6 | 95.3 | 290 | 313.3 |

*Select and ultimate, by M. A. Table. {Short men, 66 inches and under; {Men of medium height, 67 to 70 inches; {Tall men, 71 inches and over. (Page 7, Vol. II, Medico-Actuarial Investigation.)} For build classification see pp. 120-121, Vol. I, Medico-Actuarial Investigation, also p. 173, Proceedings 34th Ann. Meeting, Ass'n of Life Ins. Medical Directors, 1924.

Thirty-Fifth Annual Meeting

TABLE 3.

Comparison of Actual and Expected Mortality among Overweights, 1887-1908. By Height Class and Relative Spine Length. White Male Issues of Union Central Life Insurance Company.

BUILD CLASS I.

| BUILD CLASS AND RELATIVE SPINE LENGTH | ALL HEIGHT CLASSES | | | OVERWEIGHT MEN OF MEAN HEIGHT | | | OVERWEIGHT TALL MEN | | |
|--|--------------------|-----------------|---------------------------|-------------------------------|-----------------|---------------------------|---------------------|-----------------|---------------------------|
| | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected |
| Build class I..... | 2,786 | 2,840.9 | 98.1 | 467 | 484.4 | 96.4 | 1,687 | 1,741.5 | 96.9 |
| Relative spine length: | | | | | | | | | |
| Above limit of S. D. + from mean..... | 251 | 265.8 | 94.4 | 58 | 61.7 | 94.0 | 150 | 155.5 | 96.5 |
| Within limits of S. D. + } or — from mean..... | 2,198 | 2,224.0 | 98.8 | 361 | 369.1 | 97.8 | 1,334 | 1,360.1 | 98.1 |
| Below limit of S. D. — } from mean..... | 337 | 351.1 | 96.0 | 48 | 53.6 | 89.6 | 203 | 225.9 | 89.9 |
| | | | | | | | | | |

See Footnotes for Table 2. These apply also to this Table.

TABLE 4.

Comparison of Actual and Expected Mortality among Overweights, 1887-1908. By Height Class and Relative Spine Length. White Male Issues of Union Central Life Insurance Company.

BUILD CLASS III.

| Build Class and Relative Spine Length | All Height Classes | | | Overweight Short Men | | | Overweight Men or Medium Height | | | Overweight Tall Men | | |
|--|--------------------|-----------------|---------------------------|----------------------|-----------------|---------------------------|---------------------------------|-----------------|---------------------------|---------------------|-----------------|---------------------------|
| | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected |
| Build class II..... | 1,329 | 1,135.8 | 1.17.0 | 235 | 209.8 | 1.12.0 | 752 | 667.7 | 1.12.6 | 342 | 258.3 | 1.32.4 |
| Relative spine length: | | | | | | | | | | | | |
| Above limit of S. D. + from mean..... | 169 | 154.5 | 1.09.4 | 45 | 34.7 | 1.29.7 | 88 | 90.2 | 97.6 | 36 | 29.6 | 121.6 |
| Within limits of S. D. + or — from mean..... | 1,029 | 864.2 | 1.19.1 | 169 | 156.6 | 1.07.9 | 596 | 505.8 | 1.17.8 | 264 | 201.8 | 130.8 |
| Below limits of S. D. — from mean..... | 131 | 117.1 | 1.11.9 | 21 | 18.5 | 1.13.5 | 68 | 71.7 | 94.8 | 42 | 26.9 | 156.1 |

See Footnotes for Table 2. These apply also to this Table.

Thirty-Fifth Annual Meeting

Comparison of Actual and Expected Mortality among Overweights, 1887-1908. By Height Class and Relative Spine Length. White Male Issues of Union Central Life Insurance Company.

BUILD CLASS III.

| BUILD CLASS AND RELATIVE SPINE LENGTH | ALL HEIGHT CLASSES | | | OVERWEIGHT SHORT MEN | | | OVERWEIGHT MEN OF MEDIUM HEIGHT | | | OVERWEIGHT TALL MEN | | |
|--|--------------------|-----------------|---------------------------|----------------------|-----------------|---------------------------|---------------------------------|-----------------|---------------------------|---------------------|-----------------|---------------------------|
| | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected | Actual Deaths | Expected Deaths | Ratio, Actual to Expected |
| Build class III..... | 398 | ** | | 72 | ** | | 208 | 150.4 | 138.3 | 118 | ** | ** |
| Relative spine length: | | | | | | | | | | | | |
| Above limit of S. D. + from mean..... | 61 | ** | ** | 12 | ** | ** | 38 | 19.6 | 193.9 | 11 | ** | ** |
| Within limits of S. D. + or — from mean..... | 304 | 216.0 | 140.7 | 56 | 42.3 | 132.4 | 153 | 115.5 | 132.5 | 95 | 58.2 | 163.2 |
| Below limit of S. D. — from mean..... | 33 | ** | ** | 4 | ** | ** | 17 | 15.3 | 111.1 | 12 | ** | ** |

See Footnotes for Table 2. These apply also to this Table.

**Insufficient data.

Report of Committee on Measurements 35

MORTALITY ACCORDING TO BUILD CLASS.

The 38,062 overweight entrants gave an aggregate of 400,331 years of life exposed to risk, or an average of 10.5 years. There were 4,571 actual deaths and 4,299 expected deaths according to the M. A. Table (Select and Ultimate), or a ratio of 106.3 per cent. actual to expected mortality. Mortality ratios were 98.1 per cent. for build class I; 117.0 per cent. for build class II, and 141 per cent. for the great bulk of build class III, that is, those that were close to the average of spine length in that build class. Comparison of these risks by height shows that the overweight short men and overweight men of medium height had essentially the same mortality. Overweight tall men show in every instance higher mortality, but especially so in build classes II and III. This finding is in accordance with what has previously been found in studies of the mortality of overweights in various height groups.

MORTALITY ACCORDING TO SPINE LENGTH.

Our chief interest in this study, however, was to determine the mortality ratios in each height and build class according to the spine lengths. An examination of the tables will show that short overweights and those of medium height are different from those who are tall. For, in the first two classes, those with short spines have, in every instance, lower mortality than those that have long spines. Among tall overweights those with long spines have in every instance the better mortality. Thus, in build class I, tall men with long spines have an actual to expected mortality of 88.5 per cent., whereas those with short spines, a mortality of 120.1 per cent. There can be no mistaking the difference between these two groups. Why it is that the short men and those of medium height should behave so differently from the tall men on the score of mortality according to spine length, we do not know. This is a matter for further investigation.

Summarizing the work of the year, we may say that we have disclosed a fundamental difference between tall men on the one hand and short and medium height men on the other, in the significance of the spine lengths. In selecting tall overweights, it would appear to be indicated to choose those who are above average in spine length, that is, those who have short legs. In considering short overweights and overweights of medium height, that relationship seems to be reversed.

Thirty-Fifth Annual Meeting

Possibly, these results indicate that relative spine length is not a sufficient criterion of physical fitness among overweights. We are inclined to think that the weight of evidence points to such a conclusion. We think it will be necessary to bring into the picture not only the relative spine length of overweights, but also the chest capacity as perhaps best expressed by the mean chest measure. We purpose, therefore, if the Association will approve, to continue our investigation next year and to include the chest measure as an additional factor. We shall try to determine how spine length may be combined with chest measure to determine various degrees of physical fitness as measured by mortality.

Respectfully submitted,

OSCAR H. ROGERS,
T. H. ROCKWELL,
J. ALLEN PATTON,
LOUIS I. DUBLIN,
FANEUIL S. WEISSE,
WILLIAM MUHLBERG,
Chairman, pro tem.

Dr. Ward—Gentlemen, this subject is open for discussion. Dr. Pauli is here from the Union Central. If he has anything to say, we will be very glad to hear from him.

Dr. Pauli—Mr. President and Members of the Association—The results of the investigation do not coincide with the results that we expected. We therefore feel that since we have gone this far it would be an advantage to continue the investigation and to submit a final report next year, so that we may determine why these two groups should vary so in relation to their spine length, and in the hope that we can throw some further light on that, I think it would be well for the Association to have the Committee continue its work for another year.

Dr. Ward—This is a very interesting subject and the report of the Committee shows that they have spent a great deal of labor upon it. Are there any further remarks or questions that you would like to ask?

Dr. Dwight—Mr. President—It seems to me that the work of the Committee is very remarkable, and shows common sense as well as statistical value. I move that this report be accepted as a report of progress and the Committee asked to continue its labors.

Dr. Weisse seconded the motion, and it was carried.

Dr. Ward—The Committee appointed to review the questions on the medical blank has no report at this time to make. If there is no objection, that Committee will be continued.

On motion, duly seconded, the Committee was continued.

Dr. Ward—Gentlemen, Dr. Crawford will now present his paper on "Some Practical Observations on the Taking of Blood Pressure."

SOME PRACTICAL OBSERVATIONS ON THE TAKING OF BLOOD PRESSURE.

By G. E. CRAWFORD, PH. D., M. D.

Medical Director Cedar Rapids Life Insurance Company.

The technique of taking the blood pressure does not seem to be such a difficult procedure as to be beyond the attainment of men of ordinary intelligence. But, I think, it is the experience of every Medical Director that there is still a surprising number of examiners, some of them occupying quite high standing in the profession, who never seem to be able to acquire this art.

I recall an instance, among many others of this kind: A man who was regarded as one among the leading men of a city of fifty thousand people—and who soon after this was elected President of a State Medical Society, sent in this reading of the blood pressure of an applicant: Systolic 126—Diastolic 123 m.m. I called his attention to this impossible relation of the Diastolic and Systolic pressures, intimating that there may have been a clerical error, and asked him to please take another reading, which he sent in, with only a change of one m.m. The Systolic pressure was probably approximately correct; but it is perfectly evident that this Physician did not have the slightest conception of what the Diastolic pressure is.

What are you going to do with an examiner of this kind? This man was not really lacking in mental capacity. He would have been deeply offended at any intimation that he was not well forward in the first rank of medical prominence.

I can explain it on no other ground than downright laziness, and indifference, an easy-going satisfaction with the most superficial knowledge of things. This I believe to be the first and principal reason for unreliable blood pressure taking; just too lazy to undergo the work and pains neces-

sary to learn how to do it. Members of this class are likely to have exalted ideas of their inherent diagnostic acumen—their ability to make snap diagnoses. They boast of their ability to size up an applicant and telling whether he is a good risk by just looking him over.

To this class belong the examiners whose record cards show that the pulse is always "72-74 and the urine always has a specific gravity 1020, no albumen, no sugar." Their favorite method of analyzing the urine, is said to be, holding the bottle up to the sun, and then throwing it over the back fence.

There is another class of men who mean to be honest, but who are dull, and just naturally awkward. They always make a bungle of everything they undertake which requires any degree of manual dexterity. They do the best they can; but few of them will ever attain to any degree of proficiency in blood pressure taking. This doubtless impresses you as pessimistic, and seriously reflecting on the character of the medical profession. While these classes do exist, and all that is said about them is true, yet I hasten to say, and take great pride in saying, these classes are exceptional; that the great majority of the medical profession are honest, competent, sincere, painstaking, and faithful to every trust. But outside of incompetency and indifference, there is more or less of the inherent personal equation to be reckoned with, in taking the blood pressure, both on the part of the Doctor, and the applicant.

The original palpation method was very uncertain on this account, and is now justly obsolete. The "tactus eruditus," "the educated touch" of our forefathers, is now almost a lost art. There are persons who cannot even count the pulse accurately on account of deficiency of the tactile sense, or the absence of the sense of rhythm.

These sense deficiencies also enter into the auscultatory

method. There are many persons who are deficient in the sense of pitch, and quality of tone.

Some examiners have difficulty in recognizing the various phases, on this account, especially in recognizing the tone change at the beginning of the fourth phase, which is generally, though not universally regarded as marking the diastolic pressure. The best that such a person can do is to note the first and last sound; and he may not be able to do this with certainty.

For the reason that it is easier for many examiners to note the last sound, than the tone change, many companies direct their examiners to indicate the diastolic pressure at the disappearance of sound.

From the fact that authorities are about equally divided as to whether the diastolic pressure is located at the tone change, or the last sound, consequently making the location of the diastolic pressure very indefinite, I think it is of great importance that both these points be noted, that is the fourth and fifth points, the beginning, and ending, of the fourth phase.

Especially is this important from the fact that the fourth phase is very variable in length. The average length as shown by Dr. MacKenzie's analysis of the statistics of the Prudential Life Insurance Company, and corroborated by other statistics, is something less than six millimeters; that is, it is oftener less than six millimeters, than more than six millimeters.

We know that in many cases of high pressure, the fourth phase is only two, and three millimeters in length; while in many cases, especially of low pressure in young persons, it is ten and twenty millimeters in length, and not infrequently running down to sixty, and sometimes fifty. Of course, no one would think of stating the diastolic pressure at fifty. In these cases of very long fourth phase, the tone change can nearly always be noted, and that gives us a clue to the

location of the diastolic. It is my own opinion that the diastolic is normally located in the middle of the fourth phase, that is, about three millimeters below the tone change. At any rate, noting the tone change, and the last sound, gives us all the data there is, for locating the diastolic pressure.

I know of only two Companies who have this rule: The Equitable of Iowa, and the Cedar Rapids Life.

These Companies ask their examiners to indicate the diastolic pressure at both the fourth and fifth points, designated tone change, and last sound. Having these two points, it is up to the Medical Director to locate the diastolic pressure.

Many Companies require their examiners to give the diastolic pressure, but give them no instructions where it is located. This is the principal reason for so much faulty blood pressure taking; the absence of clear minute directions just how to take it.

It is noticed by every one who takes many blood pressure readings, that the second phase is frequently absent; that is, the harsh murmurs which are so characteristic are lacking. Also that occasionally there is a case where all the characteristic phase differentiations are absent; a tone of moderate intensity, without any change in pitch or quality, continues clear down the scale from the first to the last sound.

In cases of this kind Dr. Faught's equilibrium rule of 1, 2, 3, or rather 3, 2, 1, is of service in locating the diastolic pressure.

Now as to some of the common sources of error in taking the blood pressure: The most common of these is the effect of the procedure on the sympathetic nervous system of the applicant. Every one, these times, has heard about blood pressure. But most persons who have not seen it taken, have very vague notions concerning it. Many have the idea that it involves the drawing of blood from a vein. I am frequently asked, especially by women and boys, if it will hurt.

We know the systolic pressure is very susceptible to the reflexes of the sympathetic; the diastolic very little so. If anything abnormal is found with the blood pressure, it never should be reported from a single reading.

It is a frequent occurrence in a person with a normal, or maybe slightly raised blood pressure, to show at the first reading a systolic pressure of ten or even twenty or thirty points above what is found to be his usual pressure. In examining a subject of this sort, it should first be made clear to him that it is a perfectly simple, harmless, and painless thing, and not to pay any attention to it. It is of much importance not to allow the applicant to watch the instrument. By taking a little time, and diverting his attention, getting him to thinking about something else, after several trials, the normal point can usually be determined. With some persons it has to be deferred until another time. Some of these are persons, who cannot even have their pulse counted, without it runs up to a hundred and twenty or more. Such persons have unstable nervous systems, and are not standard risks for insurance.

There are some cases in which it is very difficult to take the blood pressure. The pulsations are almost inaudible, slight pressure with the stethoscope will render them altogether so. There may be an abnormal arrangement of the arteries. In such cases it is well to try the other arm. The phonendoscope may help some in these cases. A special clasp device to hold the instrument in place has been recommended; but I have found this a hindrance rather than a help. It is likely to cause too much pressure on the artery. An examiner will succeed best with the instrument he is in the habit of using.

The pulsations are heard louder over the brachial artery, than over the radial, just below the bifurcation; but with the wide cuffs that are now made, and the arrangement of the

inflation tubes, it is often difficult to use the brachial artery, in persons with rather short arms.

Now, what are some of the definite deductions to be made from the taking of blood pressure?

I would say that the first and most important is Dr. Fisher's deduction from his first report of the experience of the Northwestern Mutual Life Insurance Company made in 1912; and further confirmed by subsequent reports, and has been thoroughly substantiated by numerous analyses of vast volumes of experience. This deduction has become a classic in blood pressure literature, and its authority axiomatic: "A persistently high arterial tension will result in an excessive mortality; and the higher the arterial tension the greater the mortality."

I will venture this assertion, that if we could know the exact facts, the increased mortality begins with the first millimeter of increased pressure above the normal, and the mortality increases with a progressively increasing ratio, as the pressure is increased.

Just a word in regard to the significance of the various conditions found in taking the blood pressure.

High blood pressure is a very complex entity, necessarily so from the very intricate mechanism which it disturbs; and its causes are various, and not well understood; but it is essentially a toxæmia. I believe that intestinal infection, as a cause has been somewhat over emphasized, and the endocrine disturbances not sufficiently considered. Especially is this true in the so-called essential hypertension—hyperpiesis of Allbutt, with excessively high systolic pressure of 225, 250 and even 300 m.m.

The first effect of a toxic hypertension is to raise the systolic, with little or no corresponding change in the diastolic pressure.

What is the significance of a high systolic pressure with a

normal diastolic pressure, which we so often meet with? I think we are warranted in assuming that it is a recent condition, and functional, as we say, in character. That as yet no organic changes have taken place.

If we can succeed in removing the cause the equilibrium will be restored, and the patient may recover. But we may not be able to ascertain the cause, or to remove its effects.

This high pressure, at first a result, if continued will, in turn, itself become a cause; and if persistent, will produce myocardial and vascular degeneration, and interstitial nephritis, that cardio-vascular-renal cycle, which is responsible for about one-half of our mortality.

As these organic changes appear, the diastolic pressure is raised; for the diastolic is the measure of the resistance; hence its importance as the real *index* of the condition.

The diastolic pressure is more stable than the systolic, and little effected by emotion, or exercise.

When we have a high systolic pressure, with a correspondingly high diastolic pressure, we may feel practically sure that organic changes have already taken place, which can only be temporarily ameliorated and will never get well.

Probably about the only exceptions to this rule are some cases of acute nephritis, and the toxæmic nephritis of pregnancy, and they, often do not get well.

Efforts at reduction of high blood pressure should be made very carefully, or they will only result in precipitating decompensation. Or it may result in sudden myocardial weakness and death.

As yet we have no statistics of the mortality of the diastolic pressure. But I think we have sufficient knowledge of the subject to warrant us in saying that few, if any, deaths occur from high blood pressure, when there is not a high diastolic pressure.

A high diastolic pressure may be regarded as the expression of serious damage already done to vital organs.

The pulse pressure, so-called in the English-speaking profession, has no special significance of itself. It is merely a convenient term to designate the difference between the systolic and the diastolic pressures; and represents the cardiac force exerted, over and above the resistance.

What is the effect of *low* blood pressure on mortality? Again we are dependent on Dr. Fisher to answer that question. He answers it laconically by simply stating that "the experience of the Northwestern Mutual Life Insurance Company with low blood pressure is favorable."

Other Companies have had the same experience. But I believe there have been no authoritative statistics published to establish the mortality of low blood pressure.

We are not certain just what the normal blood pressure is. We know what the average blood pressure is, at the various ages. The statistics of blood pressure have a remarkable unanimity on this point. But it is altogether probable that the average blood pressure is higher than the normal blood pressure.

During the early years of the taking of blood pressure it was confined mainly to the older ages. Later many cases were taken for insurance who had a pressure considerably above the average; thus making the average blood pressure too high.

I am of the opinion that the normal blood pressure, if it can ever be determined what it is, is several points lower than the present average pressure.

I have a growing conviction, that what we call moderately low pressure, is nearer the normal, than the present tables. And I am more and more disposed to put a premium on pressure ten points below the average.

For many years I have been the official examiner for Coe College. I have approximately a thousand students to examine. Physical exercise is required of all students, unless they have physical impairments which unfit them. There is

a large Military Department, the Reserve Officers Training Corps. Athletics are much in vogue, and participated in by a large percentage of the students, Baseball, Football, Volleyball, Tennis, Basketball, and all the customary track contests. These students between the ages of 18 and 24, practically all have low blood pressure. I have examined a large number of these students for insurance, generally the older ones; many of them as fine specimens of robust health and physical perfection as Apollo ever was. And I very seldom find one with a systolic pressure above 115, and many of them 110. My explanation is that they have strong healthy hearts, and large soft blood vessels, presided over by stable nerves, and they do not need much pressure to carry on a perfectly normal circulation.

Of course, it is always imperative to eliminate tuberculosis, and all debilitating diseases as the possible cause of low blood pressure; and I believe this is usually done as carefully as any other feature of examinations.

The more I study blood pressure, and more experience I have in taking it, the more I am disposed to put a premium on low blood pressure.

AFTERNOON SESSION

Dr. Ward—Our first paper this afternoon will be presented by Dr. Harry Toulmin on the subject of "The Value of Periodic Health Examinations for Policyholders."

Dr. Toulmin—Mr. President and Gentlemen of the Association—My object in writing this paper was twofold. First, I wanted to hear from the discussion of the subject about methods of improving the service which we now offer to policyholders; and, second, I hope as the result of the discussion, that some of you who have not yet been won over to the value of the periodic health examination may be won over today. We have been offering free examinations to policyholders in the Penn Mutual at the Home Office

for some years. We have not pushed the matter in any way at all. We simply make the offer to policyholders who live in Philadelphia or nearby, in the form of a letter which we enclose with the premium receipts. Less than two years ago we offered to policyholders carrying \$25,000 minimum, an annual examination by the Life Extension Institute. The results have been wholly discouraging. We think we send a pretty good letter to the policyholders, inviting them to take this examination, and we had during eleven months in 1923 only 12% of response, and in 1924 we have had less. Of those who indicate that they would like to take the examination, 60% only have gone through with it. That is our experience. I am absolutely convinced myself of the benefits to the policyholder and to the Company, if we can only get more people to take these examinations. From those who are going to discuss this paper, I hope we may learn some way by which we can enlarge this percentage, and thus do all of us a great deal of good.

THE VALUE OF PERIODIC HEALTH EXAMINATIONS FOR POLICYHOLDERS

By HARRY TOULMIN, M. D.

Medical Director Penn Mutual Life Insurance Company.

It is quite possible that some of the members of this Association have asked the question—Why another paper on this subject so soon after the recent addresses by Doctors Knight, Muhlberg and Exton? To them my answer is three-fold: First, because our President asked me to cover this subject; second, because if it is the good thing I believe it is, it will not only stand repetition but will be aided in its development by it; third, because I am looking for help and encouragement from my fellow members.

We may dismiss the first reason by an acknowledgment of the fact that we must act as our Captain directs.

My second reason assumes that periodic health examinations are beneficial to our policyholders. Personally I not only assume this, but I am absolutely convinced of it. I know there are certain criticisms and objections to the idea as a whole and to the methods which may be employed. Doctor Exton pointed out some of them a year ago and others have called them to our attention. But to my mind, they are so insignificant when compared with the good that can be accomplished that we may justly ignore them.

I had believed in the importance of routine physical examinations long before Doctor Knight's very important and interesting address, delivered in 1921, showed such practical and satisfactory results on an economical basis. To me, and I am sure to many others, his report was most helpful in placing before other Executives the advisability of enlarging a service already in force; to others still it must have been helpful or suggestive in initiating such a service.

For several years, it has seemed to me that, wholly aside from the reason that favorable acclaim would follow the adoption of a health service and aside from the enlarged sales incident to this form of advertising, Life Insurance Companies should be pioneers in a field so little explored, so slightly known, so inadequately appreciated, not only by the people at large, but also by the Medical Profession. While it is true that today many companies offer one form of Health Service or another, it is equally true that many have as yet either failed to realize the importance of the work or have delayed putting into operation the ideas of which they have approved. Still others, feeling their way, as it were, have offered free examinations to but a small group of their membership. My own Company belongs to this class. Is it not your belief, as it is mine, that if we can get an ever increasing number of our policyholders to take routine yearly

physical examinations, it will lead to a healthier, happier and hence a larger life? It would hardly seem necessary to reiterate the many reasons why this should be so, or to enumerate the results of so many investigations where the supposedly healthy were found impaired, and the benefit arising from the proper treatment of defects and disease.

The beneficial results of routine physical examinations of our policyholders will not be confined to the improved mortality of our members, for the spreading far and wide of the need of such examinations, by old and young, rich and poor, supposedly well and probably ill, is of vast importance.

The activities of the National and State Medical Societies, of the Medical Department of the Army and Navy, and of various organizations and institutions, is one of the most encouraging signs of the day. They have not met with such a response, however, as was hoped for and anticipated. Therefore, if we can still further extend or enlarge our endeavors, it will give to all these co-workers an impetus now needed. And this leads me to my third reason for writing these few notes—my need for help and encouragement from my fellow members of the Association.

Our Health Conservation work is briefly this: For several years we have offered free examinations at our Home Office to any and all policyholders, at any time. There are a few every week. In February, 1923, we first offered an annual examination through the Life Extension Institute to all policyholders who had carried a minimum of \$25,000 for at least a year. In the eleven months of 1923, only 12% of those eligible made application and of these barely 60% took the examination.

During the first eight months of 1924, only 4.5% of those eligible made application and again about only 60% of these were examined. Our method of placing the matter before our policyholders is, I believe, similar to that of other Companies. A letter over the signature of President Law—we think for

the purpose it is very well expressed—is mailed to the eligible individuals by our General Agents; some are sent separately, others enclosed with premium notices. If he is interested, he is instructed to fill in an attached form and mail it to the Medical Director. Upon receipt of such an application, I, personally, acknowledge it and forward it to the Life Extension Institute which carries out the program from this point.

Why do so few avail themselves of our offer? Why do only 60 of each 100 who apply go through with the examination? What are the weaknesses in our method? Is the unsatisfactory outcome the result of our method, or of an inherent objection on the part of a large majority of the insured to be re-examined? Usually most people are quite ready to get something for nothing, but in this instance, the contrary is strikingly true.

If we have no response within say three months, should we send a follow up letter or letters? Were we to develop our own Conservation Department at the Home Office with a complete staff and laboratory equipment, and possibly use our own examiners for the field work, would we obtain more satisfactory results? Were we to send to all our policyholders brief leaflets on various medical topics—short and in simple language, would our results improve? Were we to extend the invitation to the much larger class of those who carry say a minimum of \$5,000 or \$10,000 in the Company, would it give us a larger percentage return?

Some of you have had a wide experience and can, I am sure, answer my questions. I feel that I want each and every one of you who has had some success in this work to give me all the suggestions possible. For, as I said early in my paper, I am absolutely convinced of the great importance of the work, the great good that can be accomplished both directly and indirectly, if only we can educate the people of our country at large, as well as our policyholders, to the importance

Discussion—Health Examinations 51

of routine examinations and the benefits to be derived therefrom.

Dr. Ward—Will Dr. Knight open the discussion of this very interesting subject?

Dr. Knight—Three years ago in my Presidential address, I reviewed the experiences of the Metropolitan on the lives of policyholders that had taken the Life Extension Institute examinations as offered and paid for by the Company. I then showed that the first 6,000 policyholders who had been examined by the Institute in the years 1914 and 1915 had during the ensuing five years shown a much lower mortality than had been expected. In fact, the saving in mortality on the A. M. (5) Table was approximately 28 per cent. The mortality was especially low even on those policyholders who on periodic examination disclosed serious impairments. These impaired lives gave a ratio of actual to expected deaths on the A. M. (5) Table of only 82 per cent. Another interesting item in our study at that time was that the greatest saving in mortality occurred in the year immediately following the Institute examination and that the saving declined from year to year until it had gone down 44 per cent in the first year to only 9 per cent in the fifth year when compared with our total Metropolitan Ordinary experience on policies issued prior to 1915.

Accordingly when President Ward asked me to discuss Dr. Toulmin's paper, it occurred to me that you would be interested to know what has transpired since the previous study was made. We have had the same 6,000 cases traced for the four more years to May 31, 1924, in order to find out whether there have been any material changes. I am able to say that following the same methods, we have found the ratio of actual to expected mortality about the same as before. There has been virtually no change in the last four years as compared with the preceding five. Taking the entire period since the examinations were made, the mortal-

ity of those policyholders on the A. M. (5) Table is only 70 per cent actual to expected. On the American Experience Table, it is only 53 per cent actual to expected.

A very interesting point is, however, raised by our Actuary which bears on this whole question of the value of periodic health examinations and shows the absolute necessity (for the sake of fairness) of continuing to compare the results or the savings from them with actual mortality experiences of the Company rather than with any expected results. He points out that during the last four years the mortality of all Metropolitan Ordinary policyholders has very materially declined from 78 per cent to 66 per cent actual to expected, on the American Experience Table. The six thousand policyholders that were examined by the Institute, on the other hand, have shown a slight increase from 59 per cent to 61 per cent in their actual to expected on the American Experience Table. In other words, the mortality in the two classes is rapidly approaching. After all, this is what we expected and predicted when we said in the address of three years ago, "The increasing scale indicates that had the experiences with those examined by the Life Extension Institute been taken for the second five years instead of for the first five years, the mortality ratio would not have been so low * * * it would therefore be safe to say that the benefits of the Life Extension examinations are pretty well limited to the years immediately following the examinations, and run out at the end of about five years * * * it would appear, therefore, that the Company has on this particular group of people had its principal returned and made a 200 per cent profit on this investment, during a period of approximately five years, provided that the subsequent experiences on these lives does not exceed the expected according to the American Men Table." The subsequent experiences during these next four years have not only exceeded that expected but have been only 74 per cent of it and only 61 per cent of the American Experience Table.

Discussion—Health Examinations 53

In the first five-year period, the saving was substantial. It was greatest in the first year after the examination. It declined appreciably in the fifth year, and in the ninth year, the experience on the examined lives was identical with that on the total Ordinary exposure (both being 69 per cent of the expected by the American Experience Table). This is not very materially different from the results we got from medical selection which wear off at the end of a five-year period. So, we, likewise, must be content with a wearing off of the effects of periodic health examination after a similar period. It would seem, therefore, that the experience has been altogether excellent for a period of nine years, but that the saving has been accomplished more particularly in the first half rather than in the second half of the period.

Dr. Ward—The Union Central has been actively engaged in this work. Will Dr. Pauli kindly continue the discussion of this paper?

Dr. Pauli—Mr. President: The subject of the Conservation of Health is of such vital importance to every member of this Association that I wish to congratulate our President for including it in the program for this meeting and we are greatly indebted to Dr. Toulmin for his valuable contribution to our Proceedings on the subject. The seed has been planted; let us nurse it and cultivate it to ripen and bear fruit many fold. The whole subject is just in its infancy and there are wonderful possibilities, and we hope that some day, every Company will be engaged in this great movement. We may have an institution endowed by the Insurance Companies to engage in research work along the lines of preventive medicine. A single discovery, such as insulin by Dr. Bantlin and his associates, would be a great contribution to our policy-holders and the nation. There is a great need for this work. We are not fatalists; we know that the law of Darwin—the survival of the fittest—is as true today as it was in 1850.

Thirty-Fifth Annual Meeting

The physicians in private practice do not appreciate the dangers of overweight at the older ages or underweight at the younger ages and their effect on mortality. We can educate the people to take an inventory of their health by a periodic examination and give them the benefit of our knowledge regarding any impairment which tends to shorten their lives, so they may be corrected before it is too late. The service we render is for a selfish motive, but nevertheless a beneficent one to humanity. The practicing physician cannot object, because we are likewise doing him a service by referring cases to him when they are amenable to treatment.

Clinics are being formed in large cities by groups of physicians with a specialist in each branch of medicine. These clinics make a practice of annual health test examinations for diagnosis only and submit a complete report of the results of their examination, similar to the Life Extension Institute of New York City. They do not accept cases for treatment. This indicates that people are beginning to appreciate the value of health test examinations. The Life Insurance Companies can spread this practice throughout the rural districts, where they have no facilities for annual health test examinations.

Dr. Muhlberg presented a paper on this subject before this Association in 1919 and gave our method of conducting our Free Health Test examination. Our policyholders continue to show an interest in this work, as is revealed in the following table:

| | | | | | |
|-------------------------------------|------|---|--------|---|---|
| In 1916 we had 11,776 Health Tests. | | | | | |
| " | 1917 | " | 20,006 | " | " |
| " | 1918 | " | 19,723 | " | " |
| " | 1919 | " | 21,408 | " | " |
| " | 1920 | " | 25,512 | " | " |
| " | 1921 | " | 26,618 | " | " |
| " | 1922 | " | 25,758 | " | " |
| " | 1923 | " | 27,043 | " | " |
| Up to Oct. 1st, 1924 " " 21,115 " " | | | | | |

Discussion—Health Examinations 55

From 1916 to October, 1924, there were almost 200,000 free health test examinations.

| | | |
|---------------------------------|---------|-------|
| O. K. (sample and health O. K.) | 95,207 | 51.4% |
| Urinary impairments | 20,568 | 11.1% |
| Sugar | 2,947 | 1.6% |
| Overweights | 7,065 | 3.8% |
| Underweights | 3,617 | 2.0% |
| Suspected Tbc. | 1,444 | 0.8% |
| Actual Tbc. | 218 | 0.1% |
| Heart Disease | 839 | 0.4% |
| Miscellaneous impairments | 53,404 | 28.8% |
| <hr/> | | |
| Total | 185,309 | |
| Additional samples examined | 13,690 | <hr/> |
| <hr/> | | |
| Grand Total | 198,999 | |

We made a total of 176 sputum tests—

| | |
|----------|-----|
| Positive | 108 |
| Negative | 66 |
| Doubtful | 2 |

Up to the present time, we have had 1970 death claims from policyholders who have submitted a Health Test. In 1923, we received 36 death claims on policyholders who died of Bright's or Diabetes, who at some time since 1915 submitted a Health Test. The analysis of these cases is shown in the following table:

| | Favorable Health Test | Unfavorable Health Test |
|-------|-----------------------|-------------------------|
| 1915— | 0 | 6 |
| 1916— | 2 | 6 |
| 1917— | 2 | 7 |
| 1918— | 1 | 9 |
| 1919— | 0 | 8 |

Thirty-Fifth Annual Meeting

| | | |
|-------|---|----|
| 1920— | 0 | 8 |
| 1921— | 0 | 16 |
| 1922— | 0 | 11 |
| 1923— | 0 | 1 |

This table gives an idea how carefully the work is done in the laboratory and that the majority of the policyholders were given a warning regarding their condition.

For our larger policyholders, on which the Company has \$25,000 or more at risk, we authorize a complete examination once a year by the Life Extension Institute. In 1923, we sent out 5,561 letters to policyholders, authorizing them to be examined by the Life Extension Institute. Of this number, 1,880 took advantage of the complete examination—about 33%.

Any Company that has a Home Office laboratory can have their policyholders submit a sample of urine to their Home Office for an annual health test examination with very little additional expense. About 12% of the policyholders will take advantage of the free health test. I can assure you it will be fascinating work and, after you have taken an interest in it, you will grow more enthusiastic over the work each year. It is a great relief to receive a letter of appreciation from a grateful policyholder, in place of a letter from an agent, complaining of the unfair treatment you have given his applicant for insurance.

Dr. Ward—I know how greatly Dr. F. C. Wells would have enjoyed this period this afternoon had he been with us. We are glad to know that although he has been removed the work in which he was so vitally interested continues. Dr. Geiringer of the Equitable will speak on this subject for that Company:

Dr. Geiringer—Gentlemen: It is a pleasure and a privilege to add a word, perhaps not of enlightenment, but at least one of commendation and appreciation for the sincerity, the enthusiasm and the conviction so evident in Dr. Toulmin's paper. The Doctor knows and he is certain that Periodic Health Ex-

Discussion—Health Examinations 57

amination is a worthy effort. Those of us who have been associated with this work are equally enthusiastic and equally certain that this comparatively new idea of Health Preservation is logical and reasonable and is particularly adapted to the general idea of Life Insurance Service.

Any physician who keeps abreast of medical progress must inevitably come to the realization that the greatest strides in medical advancement are not made along the lines of the healing art or of curative medicine, but rather in the direction of the determination of etiology, of the direct and the indirect exciting causes of diseases and their resulting physical impairments. The objective of the effort of modern medicine is to remove the cause rather than to cure the disease, to prevent the entrance of the intruder rather than to fight him when he has already entered. It is Preventive Science rather than the Healing Art.

Of course we still talk about cancer cure, tuberculosis cure, circulatory disease cure, infection cure, etc., and yet how much more sensible and how much more simple, how much more scientific, how much more practical from all standpoints is this new idea which attempts and does teach us the specific cause of diseases, the reason for the susceptibility of certain individuals to them, and the knowledge which thus places humanity in a logical position to avoid their occurrence and avert or abort their progress.

This we repeat is Preventive Medicine. The complete attainment of the goal is still very far off, and yet no close student can deny that the great strides in Life Preservation and in improved mortality have been made, not as the result of the healing art, but rather and practically entirely in the direction of disease prevention, of hygiene, of sanitation, of food study and by fortification against disease by means of vaccine and serum therapy. The startling improvement in the tuberculosis mortality of today as compared with the tuberculosis mortality of twenty-five years ago is certainly not due princi-

pally to improvement in our methods of curing this disease, but rather to our efforts in preventing its occurrence by means of education regarding exposure, food, hygiene, sanitation, etc. If the above general fact is true, and we believe that it cannot be refuted, Periodic Examination of the individual must indeed play a large part in preventive medicine. Though our methods of physical examination are still very crude we can certainly detect many forms particularly the usual forms of disease before subjective symptoms are manifest and while the disease is still in an insidious state. We are doing this every day in our experience in the Equitable Periodic Examination Service. Now, again, if it is actually true that we are detecting impairments of a character which will certainly lead to serious complications, and we assert that we are doing so, then we state the following:

Life Insurance Companies have always claimed and they must continue to claim that there is nothing more noble than a man's sacrifice during his life for the preservation of his family's material comfort after his death. Assuming that such reasoning is sound it follows as a most logical and fair conclusion that anything which will prolong or preserve this noble man's personal comfort in life, his comfort now, is also a fine effort of almost equal significance on the part of that agency which can make such preservation possible or aid it in any manner. This suggests a slogan, a method of approach—a statement of a service, which has occurred to me only a few days ago: that Life Insurance Companies may now say to their policyholders or to their prospective policyholders, "We advised you and we urged you to protect your family, your home and your business, we reminded you to protect your old age. We do all that now, perhaps more forcibly than ever and we shall continue to talk 'They' and 'For Them,' but from now on we are going to add 'you.' We are going to help you, now. We are going to improve your health and

Discussion—Health Examinations 59

prolong YOUR life for YOURSELF and therefore also for 'Them.'

Is this practical? Is this economical? Does it pay? Is it the proper function of a Life Insurance Company? Our answer is the fine and decisive statistical presentation by Dr. Knight several years ago and the further studies of the Metropolitan Life Insurance Company, the Life Extension Institute and the Equitable Life in this endeavor since that original paper. Despite the fact that statistical data is extremely difficult in this subject, we believe that it is conservative to state that irrespective of the moral duty, irrespective of the educational value to others than policyholders, irrespective of the advertising element, irrespective of the selling value and irrespective of the few objectionable possibilities, we believe that it is true that every dollar spent in this work is actually returned in far greater amount in improved mortality returns alone.

We should like to develop this subject of Periodic Examination along many lines and angles of ramification, but we were asked to comment only on Dr. Toulmin's paper. We, therefore have neither the time nor is this the place for prolonging the discussion in detail. May we answer some of his queries, however, about Life or Health Conservation work in the light of our experience?

First:

Is Periodic Examination beneficial?

Yes.

In reiteration we say nothing is finer or more altruistic than Life or Health Preservation by any agency, whether it is city, state, federal, Board of Health, Life Insurance Company or private interest.

Second:

Is Life and Health actually preserved?

Yes.

60 Thirty-Fifth Annual Meeting

Specific figures and specific cases come up daily which prove the success of a plan which is still in its infancy.

Third:

Can we justify the expense to our Companies?

Yes.

By an improved mortality which speaks dollars and cents, by the service value, by the selling value, by the advertising value and by the humane appeal of this work.

Fourth:

Why don't more policyholders accept the offer?

We believe that they do not accept the offer in as great numbers as one might expect because education is necessary; because the idea is a new one. To the average individual such an offer is too good to be true. The idea of the majority is that big business never gives anything away for nothing. It will take a long period of education and experience for the policyholder and for the company to teach policyholders that this is a sincere gift, that we shall not use any of the records which we may accumulate *against* the policyholder in his future life insurance experience. We must teach him and he must learn from experience that there is no catch in this procedure.

In conclusion may I repeat our new slogan for our present and future policyholders: "Life Insurance is a great big fine achievement for you *after* death. Periodic Health Examination is a great big fine institution for *you now*."

Dr. Ward—The next speaker is one whom many of the older members know because for many years he was a member of this Association. His name is now known throughout the civilized world because of the work he has done as Medical Director of the Life Extension Institute, and I take pleasure in introducing to this Association, Dr. Eugene Lyman Fisk, Medical Director of the Life Extension Institute.

Discussion—Health Examinations 61

Dr. Eugene Lyman Fisk—I esteem it a great honor and privilege to discuss this paper by a medical director who was not only one of the first to recognize the practical value of periodic health examinations but who has also repeatedly brought to the attention of this Association the widespread need for such a protective and corrective service. Doctor Toulmin was one of the first to call attention to the increasing menace from the chronic organic affections, in the prevention of which the periodic health examination is so effective a measure.

In his paper Doctor Toulmin shows the true scientific spirit; that is, although he is fully satisfied as to the practical value of these examinations, his paper is nevertheless a great big question mark. He is not content with having pushed a button to start this machinery, but he asks searching questions as to the sufficiency of the mechanism. Recognizing that these examinations have high value, his desire is to see the largest possible number of policyholders brought under their influence.

When your president honored me with the invitation to discuss this paper he was good enough to say that I need not confine myself to the specific questions included in the paper; that I might bring to the attention of the Association any scientific information derived from the experience of the Institute that would be of practical value. I anticipated discussing only the scientific medical aspects of this problem, but Doctor Toulmin has asked questions relating to the practical business operation of the system. I trust, Mr. President, that I may not be regarded as abusing the privilege of the floor if I attempt briefly to throw some light on these problems derived from the actual experience of the Institute in the examination of some 350,000 lives.

The principal question asked by Doctor Toulmin relates to the response of policyholders to the invitation to accept a free medical examination service. Doctor Toulmin seems

somewhat discouraged by the response of his company. But I can assure him that his returns are, as reported by him, above those of the average company. We have figured on getting about five per cent of those to whom the privilege is offered.

From my own contact with this field I am inclined to ascribe the policyholders' sluggish reaction to certain qualities inherent in human nature—which, of course, we must try to overcome. Doctor Toulmin states that most people are quite ready to take something for nothing; but I think there are many exceptions to this rule. You know the old adage, "Beware the Greeks bearing gifts!" Furthermore, these people are not getting the service for nothing, in the strict sense of the word. They must give their time and energy. They must even go to the trouble of making up their minds—and this to some people is a stupendous task. The making of the decision to have an examination of this kind, which to us seems such a commonplace affair, is to some people a very serious matter. The prosperous and thinking classes who more readily understand this message and are inclined to accept it, are busy with important affairs and their tendency is to postpone arranging for such an examination. Every month we charge off a considerable sum as a liability for people who have subscribed to our twenty-five dollar service and have failed to take the examination. There are between 1,500 and 2,000 such delinquents on our books.

Some years ago in addressing the Association of Life Insurance Presidents, Dr. Luther Gulick, a keen student of social affairs and an active social worker, stated:

"Public opinion is not the sum total of individual opinion, because public opinion is a mutually conscious opinion. Ten per cent of a community thinking alike on some topic, each one knowing that the rest are thinking alike on that topic, will create a public opinion that is powerful, much more powerful than usually obtains in any community on any subject, be-

Discussion—Health Examinations 63

cause ten per cent of any community is a large percentage to think about any one thing."

So, if Doctor Toulmin has twelve per cent of his policyholders thinking along these lines and a majority of that group actually going to the trouble of taking these examinations, I think that at the present stage of this program the result may be regarded as quite gratifying, although I agree that every possible means should be taken to further educate the policyholders and disseminate this influence more widely among them. I have been told by a great many people that they have had these examinations through their own physicians as a result of the message of the Institute.

It may be asked, why are the returns in the Penn Mutual larger than they are in other companies—for our average return, as I have said, is about five per cent. In the first place, the group is a selected one, comprising policyholders in the amount of \$25,000 or over; and there is good reason to believe from an analysis of our experience that the larger policyholders more readily accept this privilege. For example, in the Metropolitan experience, about one-half of the number examined were found in the class carrying \$5,000 policies or over. Also it was found that about one-half of the number were included in the professional and executive classes, the balance being mixed classes of the lower grades.

It is interesting to note that physicians are fairly numerous in this class but that out of every thousand applicants are found only four undertakers! It seems to me that this is good evidence that undertakers are against us; and we hope that you will love us for the enemies of that kind that we make.

Apart from the matter of class, the question raised by Doctor Toulmin as to the method of appeal may well be considered; and on this point I have some important evidence to offer.

About six months ago when the Berkshire Life Insurance

Thirty-Fifth Annual Meeting

Company was considering our service, a question was raised as to the probable number of policyholders that would take it; and assuming that a majority would avail themselves of such a valuable service, the probable expenditure involved ran into very large figures. The President of the Institute, Mr. Ley, in conference with Mr. Rhodes, vice president of the Berkshire, was quite insistent as to the improbability of the returns running beyond five per cent of the number eligible. So confident of this was he in the light of our experience that he offered to make a contract with the Berkshire Life, assuming that five per cent only of their policyholders would take the examination and that the Institute would receive the usual fee for each examination. That is, a contract was made for the examination of all policyholders in that company who would take the examination during the year for a stated sum, figured on that basis. And it has turned out that Mr. Rhodes' diagnosis was better than Mr. Ley's! We have already examined five per cent of the number to whom the privilege has been offered in that company, although the contract has only run four months. We feel, however, that the money has been well spent. Like the great surgeon who removed the man's stomach and the patient died—we have learned something.

It appears that the appeal of the Berkshire Company was a separate direct appeal to the policyholders, unaccompanied by any other communication or notice. In most companies, and in the largest companies that we serve, the appeal accompanies the premium notice. Doubtless the literature regarding the periodic health examination accompanying literature relating to the payment of premiums or other matters of insurance is often overlooked or misunderstood as having some relationship to taking out more insurance; whereas the separate appeal is more effective. I note with interest that a portion of Dr. Toulmin's policyholders received a separate invitation signed by the president of the company. This may

Discussion—Health Examinations 65

partially account for the better returns. After all, we are only in the infancy of this work. Ten years is a short time in revolutionizing the health ideals of the race. A tremendous task yet confronts us in revolutionizing the ideals in medical practice so that a full degree of co-operation may be extended by practicing physicians to people who evince an interest in prolonging their lives.

Bearing upon Doctor Toulmin's question as to whether in the course of time an increasing response from policyholders may be expected, I can present some suggestive evidence on this question.

The following table shows our experience with the Metropolitan during the past four years.

Number of Examinations by Years (A).

Ratio of Examinations to Applications by Years (B).

Total Number of Examinations from February, 1914, to end of each year (C).

Total per cent of Examinations to Applications from February, 1914, to end of each year (D).

| Year | Number Examinations by Years | Per Cent Examinations of Applications—Yearly Period | C Total | | D |
|-------------------------|------------------------------|---|--------------------------|---|---|
| | | | ined to End of Each Year | Per Cent Total Examinations of Applications | |
| 1921 | 18,413 | 73 | 68,227 | 65 | |
| 1922 | 23,950 | 67 | 94,091 | 67 | |
| 1923 | 42,477 | 67 | 134,654 | 67 | |
| to Aug. 31 | 30,157 | 70 | 167,936 | 68 | |
| 1924 est. to Dec. 31 | 49,920 | | 184,574 | | |

As a part of that experience we find a very significant fact, namely, that in New York City and its environs 95 per cent of the policyholders who apply for the privilege actually

take the examination. This extraordinary response as compared to the results throughout the country may be explained largely, I think, on the score of the greater publicity given to the Institute in New York and the fact that the policyholder is referred to a place known to be a highly organized center for rendering the service. The returns therefore from the New York section may represent the ideal toward which we are struggling throughout the country. In the course of time—in fact, plans are at present under way—similar centers in the larger cities will be established where the full standard service and follow-up of the Institute can be made available to policyholders.

The question raised by Doctor Toulmin as to whether a better response would be secured if the work were done by individual companies rather than through special organizations, I cannot appropriately discuss at any length on this occasion. But it would seem reasonable to expect that the sluggishness of the policyholder in his response to these invitations would be increased rather than diminished by the natural disinclination to have his disabilities made a matter of record in the head office of his insurance company. Furthermore, it seems to me that more publicity can be given to the work of a national institution covering a very large volume of such examinations than to the work of individual companies. However, as I said, this is a matter that I cannot discuss at length at the present time. Doctor Toulmin carried on this work at his head office before taking the service of the Institute and may have some experience on this to compare with present results.

In the chart I present showing the response of policyholders in companies representing a wide distribution territorially of this work, you will find some significant and extremely interesting figures. I have spoken of our experience with the Berkshire. I call your attention to the figures covering our work with the Union Central.

Discussion—Health Examinations 67

TABLE SHOWING RESPONSE OF POLICYHOLDERS TO OFFER OF PERIODIC HEALTH EXAMINATION BY LIFE EXTENSION INSTITUTE, AND RATIO OF EXAMINATIONS TO APPLICATIONS AND ELIGIBLES

| # | Name of Company | Years of Figures | Per Cent | Per Cent | Per Cent |
|-----|-------------------------|------------------|--------------------------------------|---|-----------------------------------|
| | | | Appli- cations of Eligibles | Examina- tions of Ap- plications | Examina- tions of Eligibles |
| P | Metropolitan (N. Y.) | 1921 | 7.8 | 69.4 | 5.4 |
| | | 1922 | | | |
| | | 1923 | | | |
| | | 1924 | | | |
| P | Guardian (N. Y.) | 1921 | 8.8 | 55.2 | 4.9 |
| | | 1922 | | | |
| | | 1923 | | | |
| | | 1924 | | | |
| P | Midland Mutual (O.) | 1922 | 7.7 | 53. | 4.1 |
| | | 1923 | | | |
| | | 1924 | | | |
| | | | | | |
| P | Fort Worth (Texas) | 1922 | * | 56. | * |
| | | 1923 | | | |
| | | 1924 | | | |
| | | | | | |
| S-P | Cedar Rapids (Ia.) | 1923 | 8.3 | 60. | 5. |
| | | 1924 | | | |
| P | Union Central (O.) | 1923 | 35. | 39. | 14. |
| | | 1924 | 36. | 47. | 17. |
| P | Mutual Life A. (Canada) | 1923 | * | 41. | * |
| | | 1924 | | 67. | |
| P | Franklin (Ill.) | 1923 | * | 7. | * |
| | | 1924 | | 46. | |
| P | Midwest (Neb.) | 1923 | 9. | 28. | * |
| | | 1924 | | 50. | |
| S | Berkshire (Mass.) | 4 mos. | | | |
| | | 1924 | 17. | 30. | 5. |

*Figures not given for eligibility.

#P Notice of eligibility sent with premium notice.

S Notice of eligibility sent in special letter at some other time.

Doctor Toulmin has asked a question as to whether educational material distributed to policyholders would be of value. In the case of the Union Central an extremely well-written and well-balanced little booklet of instruction by Dr. Muhlberg on health matters is distributed to policyholders and I should expect that such a booklet would prepare the minds of policyholders to receive an offer of this kind. You will note from the Union Central's experience that it takes a little time to get the full momentum of such service. In 1923, for example, only 14 per cent of the policyholders eligible actually took the examination. In 1924 the percentage had risen to

17 per cent. You will also note that an extraordinarily large proportion of the policyholders applied for the privilege—35 per cent in 1923, 36 per cent in 1924. This would indicate a widespread and increasing interest in the service among the policyholders of that company. Here again we have the influence of class, inasmuch as the offer was made to those holding \$25,000 policies or larger.

These figures are, of course, only approximations. It is practically impossible for any company without great expense and trouble to ascertain the exact number of individuals to whom the service is offered. In 1924, if the present rate is maintained, the number of policyholders examined during the year by the Institute will reach 75,000.

This brings me to a consideration of the scientific aspects of this service. What does it really comprise and along what lines must it develop in order to be most helpful?

I think a good deal of misunderstanding arises from using too loosely the term "periodic medical examination." Recently when in France I was told by a number of prominent health leaders and physicians that one of the great obstacles to carrying on this propaganda in France was the absolute futility of attempting to find a crisp expression in the French language that would convey a clear idea of this service. It has been said that what is not clear is not French. So we may readily appreciate how difficult it is to coin a term or expression that will not convey the notion of actual medical treatment, and still cover the procedure—the analysis of the life and body of an individual and counsel based on the findings—for the purpose of improving the quality of human life as well as extending it.

A physical examination by itself is comparatively of little value. Something must be done with the evidence elicited. This involves training minds in the interpretation of physical defects or errors in living the cumulative effect of which may mean the ultimate breakdown of health or premature death.

Discussion—Health Examinations 69

A physician who has not developed a proper concept with regard to the utilization of the evidence elicited by such examination is not, in my judgment, fully competent to conduct the examination.

This work must be conducted in a certain atmosphere of prevention—a wholly different atmosphere from that of ordinary clinical medicine or even of medical selection. The most commonplace failure of this service in general practice is where an individual goes for a health examination, has his blood pressure taken, his heart and lungs examined, and is found to be free from any well developed organic disease. In other words, he is found not to be a sick person. So he is slapped on the back and labeled a well person and sent on his way rejoicing. This, I think, is the average type of service that a layman would receive from an average physician, untaught, untrained, and wholly without stimulation or supervision from some organized center where the work has been standardized under proper ideals as to its ultimate aims.

Perhaps the most emphatic way to show the difference between the life extension and the life insurance selection attitude toward the physical examination is to consider the code symbols that are employed by the Institute for recording impairments.

I submit these codes and Hollerith cards for your records and have a supply here for distribution to the members, who are interested in them. You will note that the code covers some five hundred symbols, not all of them, of course, relating to impairments, but most of them. Many of the impairments are of a character that do not disqualify a person for life insurance and would not be of any particular interest in medical selection. But they are of practical value in constructive hygiene.

I have here a chart in which I have displayed the results elicited by the examination of groups of people under different conditions of standards, methods and supervision.

70 Thirty-Fifth Annual Meeting

COMPARISON OF THE RESULTS OF THE PHYSICAL EX-
AMINATION OF—

985 Postoffice Employees made by the United States Public Health Service;

100 Postoffice Employees examined by the Life Extension Institute;

91 Physicians (Members of the Kings County Medical Society) examined by the Committee on Dispensary Development of the United Hospital Fund; and

50 Physicians examined at the Head Office of the Life Extension Institute.

| Classes | Ratio in Each Class | | | |
|---|---|---|--|---|
| | United States Public Health Service— Post Office Employees | Life Extension Institute— Post Office Employees | Kings County Medical Society— Physicians | Life Extension Institute— Physicians |
| 1. No physical defects— | .5 | 0 | 0 | 0 |
| 2. Minor defects requiring observation or attention | 1.2 | 0 | 18.7 | 6 |
| 3. Moderate defects requiring hygenic correction or minor medical, dental or surgical attention | 26.2 | 14 | 59.3 | 20 |
| 4. Moderate defects requiring medical supervision as well as hygenic correction | 34.1 | 31 | 17.6 | 41 |
| 5. Advanced physical impairment requiring systematic medical or surgical attention | 23.8 | 43 | 4.4 | 25 |
| 6. Serious physical defects requiring immediate surgical or medical attention | 14.2 | 12 | 0 | 8 |

You will note that the United States Public Health Service and the Life Extension Institute working on similar groups of people are pretty close together in their findings. I think this is the only instance to which I can point where such close agreement exists in the results of such examinations. Before the United States Public Health Service undertook this work

Discussion—Health Examinations 71

they secured our system of classification, our medical forms, and our instructions to examiners and an effort was made to conduct the work exactly along the same lines and in the same spirit. The other two groups, while small, are nevertheless worthy of consideration.

The group of Brooklyn physicians examined were classified according to the same standards employed by the Institute; but the work was conducted and supervised by clinicians. The form employed was that of the American Medical Association, which contains nothing in the way of suggestion or detail to hold the examiner down to a comprehensive routine. This blank simply names the different regions of the body. I protested against this blank when it was submitted for my criticism on the ground that it assumed one hundred per cent diagnostic skill on the part of the examiner and one hundred per cent of interest, enthusiasm, and initiative in the matter of following up any pathological trail. The committee maintained, however, that a more extensive and complicated blank such as we employ would go over the heads, as it were, of the average practitioner; and that furthermore in the examination of supposedly well people such a searching examination was not required.

This latter judgment shows how much educational work is still necessary in the profession before a proper ideal is set up with regard to the periodic health examination service, and how necessary it is to use a medical form that sets a higher standard than is now reflected in the attitude of the general practitioner as interpreted by the American Medical Association. * * * You will observe that while many impairments were found among these Brooklyn physicians, they were mostly classified as minor in character. Possibly these physicians were self-selected on the basis of unusual enlightenment and higher ideals as to personal hygiene than prevails among aver-

*9,000 general practitioners instructed and supervised by the Institute and using these forms in a competent way, on the average.

age classes. But contrasting these results with the examination of a group of physicians at the Head Office of the Institute—not members of our staff but subscribers—usually men of a superior type, we find no such unusual condition of physical excellence as is shown in the Brooklyn group.

From a life insurance standpoint we have not looked upon physicians as in a preferred mortality class; and *a priori* there would be no reason to expect them to show superior organic condition. They lead irregular lives, are under heavy stress and nervous tension, and responsibility of life and death, and are daily called upon to do things wholly beyond the power of medical science to accomplish.

There is, of course, a very definite limit to what can be accomplished by a simple fundamental physical examination. It is merely the first step in the program for the full protection of the health of the individual. Hence the importance of making this first step in the right direction, so that subsequent steps may lead to the safety zone. The counsel based upon the results of a routine fundamental physical examination such as has been offered to life insurance companies is seldom final. It is preliminary and suggestive. The more we have studied this problem the heavier becomes our obligation not to chop off this service, as it were, abruptly with dogmatic counsel, but enlist the interest of the individual in seeking the fullest measure of protection that science can offer. This, of course, even in relation to an analysis of the individual's needs, cannot be included in the fundamental physical examination. In fully half of the cases examined there will be found need for further analysis. Merely to give to a man found to have albumin and casts in the urine the usual general explanation of such conditions and hygienic caution and counsel to go to his doctor, is unduly limiting the scope of this service. Unless such a man, and others with superficial evidence of organic pathology either present or impending, is carefully instructed with regard to the nature of the fol-

Discussion—Health Examinations 73

low-up necessary, he is likely to wander around or be supervised in a half-hearted way by a physician if he actually goes to a physician, and the service does not function with full power.

An important part, therefore, of the periodic health examination service is the setting up of standards and counsel with regard to certain groups of impairments so frequently present in the individuals examined. A policyholder fully informed in this regard is thrice armed. His report will put the physician to whom he goes for treatment on his mettle. In many cases it will suggest something to the clinician that would otherwise be neglected and it exerts a continuous educational pressure upon the practicing profession, encouraging the broader use of laboratory facilities and special methods of analysis. Time and again I have seen a letter save a life; that is, the mere suggestion as to the type of follow-up has been effective, even though made with regard to an individual three thousand miles distant, and has resulted in bringing to that individual the full resources of science.

You may ask, how is it possible with policyholders scattered all over the United States to secure a reasonable degree of co-operation and see that they get the proper follow-up. We have, of course, solved this problem at the Head Office of the Institute where these problems are pushed to the verge of the clinical field. With the growth of this work among policyholders it is a part of the Life Extension program to have similarly equipped centers in various sections of the country where the follow-up can be had in connection with the usual examinations. This is rendered possible not only by our individual work but by our work in industry which deals with large groups of employees and creates the need for highly organized facilities. Our experience has been that policyholders are very glad to avail themselves of such facilities. Having paid nothing for their examinations, the comparatively

74 Thirty-Fifth Annual Meeting

small charges for follow-up work are on the average cheerfully assumed by them. I have already referred to the fact that 95% of policyholders who apply for the service in New York actually take it.

Perhaps a reference to some of the standards employed at the Head Office of the Institute will make clear our position in this matter. In our manual of instructions to our Head Office staff we have set forth the standards to be observed in recommending the follow-up appropriate to the disabilities found in the regions examined. The policyholders in the field receive their recommendations through a reviewing staff. In cases outside of New York similar standards are observed; but at the present time and until our branch offices are established, the policyholder himself must find his own consultants and laboratories where the work can be done.

LIFE EXTENSION INSTITUTE, INC.
25 West 43rd Street,
New York City.

Statistical Analysis of Impairments found in examinations made
for
Date

CLASSIFICATION

| Class | Rating | Number | Percent |
|---|---------|--------|---------|
| No. 1 No physical defects or errors in hygiene | AA | | |
| No. 2 Minor defects requiring attention or observation. | A | | |
| No. 3 Moderate defects requiring hygienic correction, or minor medical, surgical or dental attention. | A-B & B | | |
| No. 4 Moderate defects requiring medical supervision as well as hygienic correction. | B-C | | |
| No. 5 Advanced physical impairments requiring systematic medical or surgical attention. Impairment threatening longevity. | C & C-D | | |
| No. 6 Serious physical defects requiring immediate medical or surgical attention | D & E | | |
| | Total | | 100% |

Discussion—Health Examinations 75

LIFE EXTENSION INSTITUTE, INC.

25 West 43rd St., N. Y. C.
Statistical Department

Codes for use in connection with Hollerith cards, having 45 fields with 12 code numbers to each field (including x and y as 11 and 12).

Fields 1-6 for Registration number.
" 7-9 " Group identification number.
" 10 " Number of examination (first or re-exam.)
" 11,12 " Occupation code number (see attached).
" 13 " Race and sex code number (see attached).
" 14,15 " Age.
" 16 " Weight code number (see attached).
" 17 " Height code number (see attached).
" 18 " Rating code number (see attached).
" 19 " Family History code number (see attached).
" 20-45 " Impairment code numbers (see attached).
•

CLASSIFICATION OF IMPAIRMENTS

- A.—Personal History.
- B.—Personal History.
- C.—Errors of diet and hygiene.
- D.—Effects of occupation.
- E.—Build, weight and posture.
- F.—Ears and eyes.
- G.—Nose and throat.
- H.—Teeth and root infection.
- I.—Heart and pulse.
- J.—Blood-vessels and blood-pressure.
- K.—Lungs and other respiratory.
- L.—Stomach and abdominal organs.
- M.—Abdominal organs and inguinal region.
- N.—Genito-urinary.
- O.—Brain and nervous system.
- P.—Endocrine disturbances.
- Q.—Miscellaneous impairments.
- R.—Miscellaneous impairments.
- S.—Urinalysis.
- T.—Urinalysis, continued, and blood urea.
- U.—Special tests.
- V.—Special tests.
- W.—Tumors, benign or malignant, found or suspected.
- X.—X-rays made.
- Y.—X-ray of heart and chest.

Thirty-Fifth Annual Meeting

Life Extension Institute—Statistical Dept.
OCCUPATION CLASSIFICATION
Alphabetically Arranged.

| | |
|--|---------------------------------------|
| 49. Accountants and auditors. | ers. |
| 67. Actors and public entertainers. | 15. Furriers. |
| 40. Agents. | 14. Garment operatives. |
| 1. Agricultural workers. | 46. Government officials. |
| 50. Architects and draftsmen. | 97. Granite workers. |
| 51. Artists. | 1. Horticulturists. |
| 52. Authors. | 65. Hotel keepers. |
| 16. Bakers. | 69. Housewives. |
| 71. Bankers. | 83. Inspectors. |
| 64. Barbers. | 12. Iron workers (structural). |
| 18. Blacksmiths. | 20. Iron workers. |
| 84. Boarding house keepers. | 113. Janitors. |
| 68. Bookkeepers. | 27. Jewelers and silversmiths. |
| 22. Brick, pottery, terra cotta, and tile makers. | 52. Journalist. |
| 7. Bricklayers. | 92. Laborers. |
| 71. Brokers. | 115. Laundry employees. |
| 4. Builders. | 58. Lawyers. |
| 82. Butchers. | 23. Leather Products makers. |
| 41. Buyers and purchasing agents. | 94. Librarian. |
| 5. Carpenters. | 30. Lithographers. |
| 72. Cashiers. | 21. Machinists (office, store). |
| 86. Chauffeurs. | 70. Managers (plant). |
| 53. Chemists. | 81. Manufacturers. |
| 54. Clergymen. | 39. Marine officers. |
| 68. Clerks. | 7. Masons. |
| 14. Clothing operatives. | 4. Mechanics (master). |
| 40. Collectors. | 42. Merchants and jobbers. |
| 44. Commissioned officers of the Army and Navy. | 88. Metal workers. |
| 4. Contractors. | 99. Milliners. |
| 87. Cutters. | 3. Miners (all). |
| 55. Dentists. | 90. Missionaries. |
| 51. Designers. | 91. Musicians. |
| 96. Domestic help. | 59. Nurses (trained). |
| 98. Dressmakers. | 73. Officers of corporations. |
| 111. Drivers (not chauffeurs). | 60. Optometrists. |
| 56. Druggists. | 13. Paint and pigment makers. |
| 52. Editors. | 8. Painters. |
| 6. Electricians. | 9. Paperhangers. |
| 114. Elevator operators. | 29. Paper products makers. |
| 57. Engineers (civil, mechanical, electrical, mining). | 116. Peddlers. |
| 36. Engineers (stationary). | 31. Photoengravers. |
| 93. Factory workers, unclassified (light). | 74. Photographers. |
| 36. Firemen. | 61. Physicians and surgeons. |
| 45. Firemen (city department). | 10. Plasterers. |
| 2. Fishermen. | 11. Plumbers, pipe and steam fitters. |
| 17. Food canners and preservers. | 47. Police officers. |
| 19. Foundry workers. | 48. Post Office employees. |
| 25. Furniture and cabinet mak- | 62. Principals. |
| | 32. Printers. |
| | 62. Professors. |
| | 33. Publishers. |
| | 37. Railroad employees. |

Discussion—Health Examinations

77

| | |
|---|-----------------------------------|
| 65. Restaurant keepers. | 62. Teachers. |
| 112. Reporters. | 76. Telegraph operators. |
| 80. Retired. | 76. Telephone operators. |
| 35. Rubber mill employees. | 72. Tellers. |
| 43. Salesmen. | 34. Textile mill operators. |
| 24. Shoe factory operatives. | 28. Tinsmiths. |
| 40. Solicitors. | 77. Undertakers and embalmers. |
| 20. Steel mill workers. | 89. Upholsterers. |
| 117. Stewards. | 13. Varnish and dry color makers. |
| 38. Street railway employees. | 63. Veterinarians. |
| 75. Students. | 66. Waiters and hotel servants. |
| 70. Superintendents (plant, office, store). | 95. Watchmen. |
| 62. Superintendents (school). | 26. Woodworkers. |
| 85. Tailors. | 52. Waiters (professional). |
| | 99. All others. |

STANDARD FOR NORMAL BLOOD PRESSURE

| Average Systolic | Pressure | Millimeters |
|------------------|----------|-------------|
| Ages 15 to 20 | — | 120 |
| " 21 to 25 | — | 123 |
| " 26 to 30 | — | 124 |
| " 31 to 35 | — | 124 |
| " 36 to 40 | — | 127 |
| " 41 to 45 | — | 129 |
| " 46 to 50 | — | 131 |
| " 51 to 55 | — | 132 |
| " 56 to 60 | — | 135 |

RACE AND SEX

| | | |
|--------------------|----------------------|----------------|
| 1. White male. | HEIGHT | RATING |
| 2. White female. | 0. No height given. | 0. None given. |
| 3. Colored male. | 1. Under 4' 8" | 1. AA |
| 4. Colored female. | 2. 4' 8" + to 4' 10" | 2. A |
| 5. | 3. 4' 10" + to 5' | 3. A-B |
| 6. | 4. 5" to 5' 2" | 4. B |
| 7. | 5. 5' 2" to 5' 4" | 5. B-C |
| 8. | 6. 5' 4" + to 5' 6" | 6. C |
| 9. | 7. 5' 6" + to 5' 8" | 7. C-D |
| 10. | 8. 5' 8" + to 5' 10" | 8. D |
| 11. Born in U. S. | 9. 5' 10" + to 6' | 9. D-E |
| 12. Foreign born. | 11. 6' + to 6 2" | 11. E |
| | 12. Over 6 2" | 12. |

WEIGHT%

| | |
|--|--|
| 0. None given. | FAMILY HISTORY |
|1. Over 40% underweight | 0—None given. |
|2. 40—30% " | 1—Tuberculosis. |
|3. 30—20% " | 2—Cancer. |
|4. 20—10% " | 3—Epilepsy. |
|5. 10— 5% " | 4—Nephritis or Bright's disease. |
|6. Normal (less than 5% under- or over-weight) | 5—Apoplexy or arterial disease, stroke, cerebral hemorrhage. |
|7. 5—10% overweight | 6—Nervous diseases. |
|8. 10—20% " | 7—Insanity. |
|9. 20—30% " | 8—Other pulmonary diseases than tuberculosis. |
|11. 30—40% " | 9—Syphilis. |
|12. Over 40% " | 11—Diabetes. |
| | 12—General lack of longevity. |

Thirty-Fifth Annual Meeting

A.—PERSONAL HISTORY 1—Complaint of fatigue (lack of energy).

0—Normal (none given). 2—Hours of work too long.
 1—Tuberculosis. 3—Lack of exercise.
 2—Typhoid fever. 4—Occupational poisoning.
 3—Influenza (recent). 5—“ strain.
 4—Gall-stones, or gall-bladder disease. 6—Exposure to great heat.
 5—Renal calculi (kidney stones). 7—“ “ poisonous fumes.
 6—Nephritis (Bright's disease). 8—“ dust.
 7—Epilepsy. 9—“ excessive moisture.
 8—Nervous breakdown. 11—“ noise.
 9—Miscarriage (spontaneous) or stillborn. 12—Eye strain.

11—Neuritis.

12—Ulcers of stomach or intestines or other severe gastric disturbances.

B.—PERSONAL HISTORY

0—None. 1—Light frame.
 1—Operation for hernia. 2—Heavy “
 2—Operation for appendicitis. 3—Recent loss in weight.
 3—History of cancer. 4—
 4—Operation for condition in pelvic region. 5—
 5—Rheumatism or gout (muscular or arthritis). 6—
 6—Tonsilitis. 7—
 7—Pleurisy. Pneumonia or other pulmonary infection (not T.B.) 8—Faulty posture.
 8—History of removal of tumor, breast, uterus. 9—Spinal curvature.
 9—Gonorrhea (old infection). 11—Flat foot.
 11—Syphilis (positive). 12—Bony deformities, ankylosis, and subtraction.

C.—ERRORS IN DIET AND HYGIENE

0—None found. 0—Normal.
 1—Too high protein diet. 1—Defective vision—corrected.
 2—Too little water consumed. 2—“ “ —uncorrected.
 3—Too much tea and coffee. 3—Disease of external eye or eyelids.
 4—Other errors in diet. 4—Eye ground changes.
 5—Alcohol temperate. 5—Blindness in both eyes.
 6—“ excessive. 6—Cataract.
 7—Tobacco temperate. 7—Defective hearing.
 8—“ excessive. 8—Practically total deafness.
 9—Rapid eating. 9—Otitis media or discharging ears.
 11—Diet deficiency (specific vitamin—bulk insufficient). 11—Perforation of drum with or without operation.
 12—Diet excess (general). 12—Wax in ears.

D.—EFFECTS OF OCCUPATION

0—None.

E.—BUILD, POSTURE, WEIGHT

0—Normal. 1—Light frame.
 1—Light frame. 2—Heavy “
 2—Heavy “ 3—Recent loss in weight.
 3—Recent loss in weight. 4—
 4—
 5—
 6—
 7—
 8—Faulty posture.
 9—Spinal curvature.
 11—Flat foot.
 12—Bony deformities, ankylosis, and subtraction.

F.—EYES AND EARS

0—Normal. 1—Defective vision—corrected.
 1—Defective vision—corrected. 2—“ “ —uncorrected.
 2—“ “ —uncorrected. 3—Disease of external eye or eyelids.
 3—Disease of external eye or eyelids. 4—Eye ground changes.
 4—Eye ground changes. 5—Blindness in both eyes.
 5—Blindness in both eyes. 6—Cataract.
 6—Cataract. 7—Defective hearing.
 7—Defective hearing. 8—Practically total deafness.
 8—Practically total deafness. 9—Otitis media or discharging ears.
 9—Otitis media or discharging ears. 11—Perforation of drum with or without operation.
 11—Perforation of drum with or without operation. 12—Wax in ears.

G.—NOSE AND THROAT

0—Normal. 0—Normal.
 1—Deflected septum—slight. 1—Deflected septum—slight.
 2—“ “ —marked. 2—“ “ —marked.
 3—Enlarged, septic or buried tonsils. 3—Enlarged, septic or buried tonsils.
 4—Naso-pharyngitis (chronic). 4—Naso-pharyngitis (chronic).
 5—Naso-pharyngitis (acute). 5—Naso-pharyngitis (acute).
 6—Adenoids. 6—Adenoids.

Discussion—Health Examinations 79

| | |
|--|--|
| <p>7—Infection of nasal accessory sinus.</p> <p>8—Hypertrophic rhinitis (enlarged turbinates).</p> <p>9—Atrophic rhinitis.</p> <p>11—Perforation of septum (no history of operation).</p> <p>12—Polypi, growths, ulcers.</p> <p>H.—TEETH AND ROOT INFECTION</p> <p>0—Normal.</p> <p>1—Carious teeth, septic roots, need dental attention.</p> <p>2—Slightly infected gums (including recession).</p> <p>3—Fyorrhea (definite).</p> <p>4—Heavy dentistry (X-ray advised, suspected infection).</p> <p>5—X-ray shows infection of one tooth.</p> <p>6—X-ray shows infection of two or more teeth.</p> <p>7—X-ray shows resorption of alveolar process.</p> <p>8—Insufficient dentistry (missing teeth).</p> <p>9—Irregular and maloccluded teeth.</p> <p>11—Root infection with systemic symptoms (Anemia, Rheumatism, L. B. P.)</p> <p>12—Root infection with organic symptoms (Heart, arteries, lungs, H. B. P.)</p> <p>I.—HEART AND PULSE</p> <p>0—Normal.</p> <p>1—Functional murmur or irregularity.</p> <p>2—Mitral murmur—stenosis.</p> <p>3—Mitral murmur—regurgitation (leaking valve).</p> <p>4—Aortic murmur—stenosis.</p> <p>5—Aortic murmur—regurgitation (leaking valve).</p> <p>6—Enlargement.</p> <p>7—Myocardial changes.</p> <p>8—Slow pulse below 58.</p> <p>9—Rapid pulse above 90.</p> <p>11—Intermittent pulse, extra systoles.</p> <p>12—Auricular fibrillation, heart block, paroxysmal tachycardia or other serious impairments including murmurs of tricuspid valve.</p> | <p>J.—BLOOD VESSELS AND BLOOD PRESSURE</p> <p>0—Normal condition of blood vessels.</p> <p>1—Arterial thickening, slight.</p> <p>2—“ “ moderate.</p> <p>3—“ “ marked.</p> <p>4—Other blood vessel changes.</p> <p>5—Varicose veins.</p> <p>6—Normal blood pressure.</p> <p>7—B. P. 15-25 below average for age.</p> <p>8—B. P. 25 or more below average for age.</p> <p>9—B. P. 20-40 above average for age.</p> <p>11—B. P. 40-60 above average for age.</p> <p>12—B. P. 60 or more above average for age.</p> <p>K.—LUNGS AND OTHER RESPIRATORY</p> <p>0—Normal.</p> <p>1—Bronchitis (acute).</p> <p>2—“ (chronic).</p> <p>3—Asthma.</p> <p>4—Emphysema.</p> <p>5—Abnormal signs in lungs, not suggestive of T. B.</p> <p>6—Suspected tuberculosis.</p> <p>7—Incipient “</p> <p>8—Moderately advanced tuberculosis.</p> <p>9—Advanced tuberculosis.</p> <p>11—</p> <p>12—Sputum analysis made.</p> <p>L.—STOMACH AND ABDOMINAL ORGANS</p> <p>0—Normal.</p> <p>1—Acid stomach.</p> <p>2—Gastric disturbances.</p> <p>3—Constipation.</p> <p>4—Diarrhea.</p> <p>5—Tenderness over liver or gall bladder.</p> <p>6—Enlargement or hardening of liver.</p> <p>7—Tenderness in region of appendix.</p> <p>8—Ulcer of stomach or intestines, found or suspected.</p> <p>9—Hemorrhoids, or bleeding from rectum.</p> <p>11—Fistula in ano.</p> <p>12—Fissure in ano, ulcers.</p> |
|--|--|

80 Thirty-Fifth Annual Meeting

| | |
|---|--|
| <p>M.—ABDOMINAL AND INGUINAL REGIONS</p> <p>0—Normal. 1—Weak inguinal rings. 2—Inguinal hernia no truss. 3— " truss worn. 4—Other hernias. 5—Gastro enteroptosis, viscerop-tosis. 6—Varicocele. 7—Hydrocele. 8— 9— 11— 12—</p> <p>N.—GENITO-URINARY</p> <p>0—Normal. 1—Prostate enlarged, hard, tender or boggy. 2—Stricture of urethra. 3—Testicles hard or tender. 4—Frequent or painful urination (nocturia). 5—Cystitis (inflammation of bladder). 6—Tenderness over ovary. 7—Rectocele or cystocele. 8—Severe displacement or dis-eases of uterus (laceration of cervix). 9—Dysmenorrhea, leucorrhea, profuse or irregular menstrua-tion. 1—Syphilitic lesion or sores—or doctor's positive statement of syphilis (lues). 12—</p> <p>O.—BRAIN AND NERVOUS SYSTEM</p> <p>0—Normal. 1—Nervousness. 2—Exaggerated reflexes and tre-mors not due to nervousness. 3—Reflexes sluggish, absent, un-equal or irregular. 4—Ataxic speech or gait. 5—Romberg—positive. 6—Cerebro-spinal syphilis. 7—Neurasthenia, psychasthenia, excitability (hysteria), de-pression (melancholia). 8— 9— 11—Nervousness with increased reflexes. 12—Neurological or neuropsychi-atric examination made.</p> | <p>P.—ENDOCRINE DISTUR-BANCES</p> <p>0—Normal. 1—Hypothyroidism. 2—Enlarged thyroid—simple goitre. 3—Evidence of dysfunction of thyroid (toxic) thyroid or ex-ophthalmic goitre. 4—Evidence of dysfunction of pituitary gland. 5— 6—Adrenal deficiency. 7—Pluriglandular dysfunction. 8—Gonadal dysfunction (ovaries or testicles). 9— 11— 12—Basal Metabolism test made.</p> <p>Q.—MISCELLANEOUS IM-PAIRMENTS</p> <p>0—Normal. 1—Adenitis (glands, lymphatics). 2—Anaemia—hemoglobin below 75. 3—Tuberculosis of glands or other than pulmonary. 4—Chronic skin affection (acne, Dermatitis, eczema, pruritis ani). 5—Marked pigmentation. 6—Dizziness (vertigo). 7—Insomnia. 8—Backache. 9—Headache. 11—Use of patent medicine. 12— " laxatives.</p> <p>R.—MISCELLANEOUS IM-PAIRMENTS</p> <p>0—Normal. 1—Laryngitis chronic. 2— 3—Frequent colds. 4—Mastoids—History of or found at present time. 5—Oedema. 6—Neuralgia or neuritis. 7—Paralysis of any degree or place. 8—Not aware of impairments found. 9—Aware of impairments found. 11—Now under doctor's care. 12—Unclassified impairment.</p> |
|---|--|

Discussion—Health Examinations 81

S.—URINALYSIS FINDINGS

- 0—Normal.
- 1—Albumin, slight trace.
- 2— " definite trace.
- 3— " marked amount.
- 4—Sugar, trace.
- 5— " marked amount (quantitative).
- 6—Acetone.
- 7—Pus (large amount or persistent), (numerous leucocytes).
- 8—Blood.
- 9—Casts (granular or epithelial).
- 11— " (hyaline).
- 12—Indican.

- 3—Vaginal smear not showing gonorrhreal infection.
- 4—Vaginal smear showing gonorrhreal infection.
- 5—Wassermann negative.
- 6—Second Wassermann negative.
- 7—Wassermann slightly positive 1 or 2 + plus (+)
- 8—Wassermann strongly positive, 3 or more + plus (+)
- 9—Second Wassermann positive.
- 11—Gastric analysis—normal.
- 12— " " HCl. reduced or increased—shows blood or other pathology.

T.—URINALYSIS FINDINGS

AND BLOOD UREA FINDINGS—Continued

- 0—Normal.
- 1—Low specific gravity.
- 2—High "
- 3—24-hour quantitative.
- 4—Concentration test.
- 5—Sodium chlorides test.
- 6—complete blood chemistry.
- 7—Minerals significantly changed.
- 8—Renal efficiency test.
- 9—
- 11—Urea nitrogen, blood test.
- 12—Uric acid, blood test.

W.—CYSTS, TUMORS, CANCERS

Split Column

- 0—None found.
- 1—Head.
- 2—Breast.
- 3—Stomach.
- 4—Intestines.
- 5—Uterus.
- 6—Ovaries.
- 7—Skin (epitheleoma, moles).
- 8—Fatty.
- 9—Other part of body.
- 11—Probably benign.
- 12— " malignant.

U.—SPECIAL TESTS

- 0—None made.
- 1—Feces analysis.
- 2—
- 3—Special rectal examination.
- 4—Complete blood count.
- 5—Differential white blood count.
- 6—Special heart examination.
- 7—Sugar tolerance test normal.
- 8— " " abnormal.
- 9—Blood sugar test normal.
- 11— " " abnormal.
- 12—Tonsil culture.

X.—X-RAYS MADE, PROTEIN SENSITIZATION TEST

- 0—None made.
- 1—Teeth.
- 2—Chest, lungs, heart.
- 3—Head.
- 4—Spine.
- 5—Legs, arms and joints.
- 6—Gastro-intestinal.
- 7—Kidneys.
- 8—Gall-bladder region.
- 9—Protein sensitization test negative.
- 11—Protein sensitization test positive epidermal or for pollen.
- 12—Protein sensitization test positive for food or bacteria.

V.—SPECIAL TESTS

- 0—None made.
- 1—Prostatic smear not showing gonorrhreal infection.
- 2—Prostatic smear showing gonorrhreal infection.

Thirty-Fifth Annual Meeting

| Y.—X-RAY OF HEART AND CHEST | Z.— |
|--|---|
| 0—Chest, heart and lungs negative or normal. | 0—No improvement. |
| 1—Heart enlarged—slightly. | 1—Improvement in diet or hygiene. |
| 2— " " definitely. | 2—Improvement in physical condition. |
| 3— " displaced. | 3—Tonsils removed, improvement noted. |
| 4— " sinuses infected. | 4—Tonsils removed, no improvement noted. |
| 5— " shows dilated aorta, aortic changes. | 5—Infected teeth removed, improvement noted. |
| 6—Circulation—aneurysm. | 6—Infected teeth removed, no improvement noted. |
| 7— | 7— |
| 8—Lungs show marked bronchial thickening. | 8— |
| 9—Lungs show old foci of tuberculosis. | 9— |
| 11—Lungs show active foci of tuberculosis. | 11—Check-up (mid-year P. H.) |
| 12— | 12—Check-up—Examination. |

I present herewith standardized instructions as to laboratory tests and X-ray or special regional examinations considered necessary or advisable as supplementary investigations in a complete physical examination:

THE MOUTH

(Findings or history grouped under tests to simplify outline)

X-ray of the Teeth and Jaws indicated:

1. In all cases of pulpless teeth, decay or repairs.
2. After complete extraction, to determine presence of infected root particles.
3. History of facial neuralgia, malposed, impacted, and supernumerary imbedded teeth suspected as a cause.
4. Tumors of jaw.
5. Routine, history of rheumatism, neuritis, evidence of focal infection.

Wasserman Blood Test indicated—

1. Ulcerations, mucous patches, leukoplakia, gummatous indurations, and perforations.
2. Jaw tumors.

Microscopy indicated—

Ulcerations, exudations, membranes.

Cultures indicated—

Ulcerations, exudations, membranes.

X-ray of Lungs indicated—

When ulcerations appear to be tuberculous.

Other conditions call for special tests; as, for example: excessive pigmentation of the soft palate—blood sugar determination; pallor of the tongue and glossitis—complete blood count; petechial hemorrhages—blood cultures; the lead line—examination of the red cells for stippling, and chemical examination of the urine; atrophies involving the tongue or the taste buds—the Wassermann reaction.

THE NOSE AND THROAT

(Findings or history grouped under tests to simplify outline)

X-ray of the Sinuses indicated—

1. When physical findings suggest present trouble.
2. History of sinusitis.
3. History of focal infection when teeth and tonsils have been excluded.

Tonsil Culture indicated—

1. When tonsils present suspicious appearance.
2. History of repeated attacks of tonsillitis.
3. History rheumatic affections or rheumatic fever.
4. Cardio-renal disturbance.

Protein Sensitization Test indicated—

Where there are symptoms or history of hay fever.

THE EYE

(Tests grouped under findings or history to simplify outline.)

Pupils fixed or sluggish to light, unequal or irregular—

1. Blood Wassermann Test.
2. Wassermann Spinal Fluid Test.

Thirty-Fifth Annual Meeting

Fundus showing signs of chronic choroiditis or retinitis, not readily explained as to etiology—

1. Blood Wassermann Test.
2. Wassermann Spinal Fluid Test.

Retinitis, fresh or old hemorrhages, other abnormality of kidney origin—

Investigation of Kidney Function (Blood Chemistry, Urine Concentration, Dye Test).

Diabetic retinitis—

1. Blood Chemistry.
2. Urine for Sugar.

Sclerotic retinal vessels—

1. Kidney Function Tests.
2. Blood Wassermann Test.
3. X-ray of Teeth.
4. X-ray of Sinuses.
5. Tonsil Culture.

Iritis—

1. Blood Wasserman Test.
2. X-ray of Teeth.
3. X-ray of Sinuses.
4. Tonsil Culture.

THE EAR

(Tests grouped under findings or history to simplify outline)

Chronic suppurative otitis—

1. This condition is often indicative of more or less involvement of the mastoid cells (chronic mastoiditis) and should be further investigated by X-ray Examination. In this connection it is important to remember that the mastoid area, both right and left, should be taken for the purpose of comparison.

Discussion—Health Examinations 85

2. Purulent discharges from the middle ear should be spread upon glass slides, stained, and examined by the microscope. Culture should also be taken.

Disturbances of the vestibular apparatus—

1. Functional Tests.
2. Blood Wassermann Test.

In all cases of deafness where the cause is not apparent—

Blood Wassermann Test.

THE CIRCULATORY SYSTEM

(Tests grouped under findings or history to simplify outline)

Valvular disease of heart—

1. X-ray of Heart.
2. Blood Wassermann Test.
3. Blood Culture (if temperature).
4. Kidney Function Tests.
5. Search for Focal Infection (X-ray of Teeth, X-ray of Sinuses, Tonsil Culture, etc.).

Displaced heart—

1. X-ray of Heart.
2. X-ray Gastro-intestinal Tract.
3. Blood Wassermann Test.

Asthma—

1. X-ray of Heart and Chest.
2. Electrocardiogram.
3. Search for Focal Infection.
4. Protein Sensitization Test.
5. Kidney Function Tests.
6. Blood Wassermann Test.

Angina Pectoris—

1. X-ray of Heart.
2. Electrocardiogram.

86 Thirty-Fifth Annual Meeting

3. Search for Focal Infection (X-ray of Teeth, X-ray of Sinuses, Tonsil Culture, Gastro-intestinal X-ray, Prostatic Massage and Smear).
4. Kidney Function Tests (if high blood pressure).
5. Blood Wassermann Test.

Irregularities—

1. Electrocardiogram.
2. X-ray of Heart.
3. Search for Focal Infection (X-ray of Teeth, X-ray of Sinuses, Tonsil Culture, Gastro-intestinal X-ray).
4. Blood Wasserman Test.

High blood pressure—

1. X-ray of Heart.
2. Electrocardiogram.
3. Search for Focal Infection (X-ray of Teeth, X-ray of Sinuses, Tonsil Culture, Gall Bladder X-ray, Gastro-intestinal X-ray).
4. Kidney Function Tests.
5. Blood Wasserman Test.

Low blood pressure—

1. Search for Focal Infection (X-ray Teeth, X-ray Sinuses, Tonsil Culture, Gastro-intestinal X-ray, Prostatic Massage and Smear).
2. Blood Wassermann Test.
3. Blood Count.
4. X-ray of Chest (if underweight).
5. Sputum Examination.
6. Basal Metabolism (if overweight).

Vascular thickening—

1. Search for Focal Infection.
2. Kidney Function Tests.
3. Electrocardiogram.
4. Blood Wassermann Test.

Aneurisms—

1. X-ray of Heart and Lungs.
2. Blood Wassermann Test.
3. Gastro-intestinal X-ray (if abdominal).

Endarteritis—

1. Blood Wassermann Test.
2. Search for Focal Infection.
3. X-ray of vessels.

THE RESPIRATORY SYSTEM

(Tests grouped under findings or history to simplify outline)

Upper tract—

(See Nose and Throat.)

Asthma—

1. X-ray of Chest and Heart.
2. Protein Sensitization.
3. Blood Chemistry.
4. Urine Concentration.
5. Special Heart Examination (if circulation shows any sign of disturbance).

Suspected Tuberculosis—

1. X-ray of Chest (including fluoroscope).
2. Numerous Sputum Analyses.
3. Complete Blood Count.
4. Observation by Physician at Short Intervals.

History of influenza or pleurisy—

X-ray of chest and observation.

ABDOMINAL ORGANS AND DIGESTIVE SYSTEM

Symptoms suggestive of abnormalities of oesophagus should be investigated by X-ray, using both fluoroscopic and radiographic methods after ingestion of opaque paste. When indicated investigation should include—

1. Examination with Oesophagoscope.
2. Microscopic Examination of Tissue.
3. Blood Wassermann.

(Tests grouped under common symptoms, findings or history
to simplify outline)

Indigestion, dyspepsia, epigastric pain, hematemesis—

1. Gastria Analysis.
2. Gastro-intestinal X-ray.
3. Feces for Blood.
4. Blood Wassermann.
5. X-ray of Chest.
6. Blood Count.

Abdominal pain—

1. Gastro-intestinal X-ray.
2. Blood Wassermann.
3. X-ray Gall Bladder.
4. X-ray Kidney, Ureters and Bladder.
5. X-ray Appendix (opaque enema).
6. Feces for Blood, undigested food, azotorrhea, steatorrhea, parasites.
7. Feces screened for Billary Calculi.
8. Urine for Blood and Bacteria.
9. Blood Count.

Nausea and vomiting—

1. Gastro-intestinal X-ray.
2. Gall Bladder X-ray.
3. Blood Wassermann.
4. Blood Chemistry.
5. Urine for Indican, Albumin, etc.
6. Blood Count.

Constipation, Diarrhoea—

1. Gastro-intestinal X-ray.
2. Blood Count and Hemoglobin.
3. X-ray of Colon after opaque enema.

Discussion—Health Examinations 89

4. Feces for Blood and Parasites.
5. Sigmoidoscopy and Proctoscopy.

Melena—

1. Gastro-intestinal X-ray.
2. Sigmoidoscopy and Proctoscopy.
3. X-ray of Colon after opaque enema.
4. Complete Blood Count.
5. Feces for Parasites, Blood, etc.

Jaundice—

1. Gastro-intestinal X-ray.
2. Gall Bladder X-ray.
3. Complete Blood Count.
4. Feces.
5. Urine for Bile.

Abdominal Tumor—

1. Re-examination after thorough catharsis.
2. Gastro-intestinal X-ray.
3. Sigmoidoscopy.
4. Blood Wassermann.
5. Complete urinary X-ray.
6. Blood Count.

Splenomegaly—

1. Complete Blood Count.
2. Search for Parasites.
3. Blood Wassermann.

ANUS AND RECTUM

History of bleeding, pruritis, or other symptom suggesting local disturbance—

Special Protoscopic examination and X-ray of the Colon in appropriate cases.

Thirty-Fifth Annual Meeting

GENITO-URINARY SYSTEM

(MEN)

(Tests grouped under symptoms, history or findings to simplify outline)

Suspected impairment of kidney function—

1. Urine Concentration Test.
2. Blood Chemistry.
3. Dye Test.
4. Periodic analyses.
5. (For women) Catheterized specimen.

Suspected calculi in kidneys, bladder, ureters, vesicles, unexplained hematuria, diverticula—

1. X-ray of region.
2. Cystoscopy.

Suspected tuberculosis—

1. Pyelography.
2. Cystoscopy.
3. Special microscopic examination of urine sediment.

Suspected or found tumors of kidney or prostate—

1. X-ray of region.
2. Pyelography (if in kidney).
3. Cystoscopy (if in prostate).

Urethral discharge, present or history—

1. Repeated Urinalyses.
2. Microscopic Examination of discharge or
3. Material obtained after Prostatic Massage.

Ulceration—

Microscopic examination of scrapings.

Chancre suspected—

Dark Field examination of material.

History of syphilis or findings suggestive of syphilis—

Blood Wassermann Test.

Discussion—Health Examinations 91

History of Gonorrhea—

1. Blood Wassermann Test.
2. Gonococcus Fixation if active infection is suspected.

Sterility—

Examination of seminal fluid of man (specimen to be collected and kept at body temperature according to instructions).

GENITO-URINARY SYSTEM (WOMEN)

Same as for men for urinary apparatus.

Leucorrhœal discharge, intermenstrual bleeding, foul odor, pain call for—

1. Repeated gynecological observation.
2. Microscopic Examination of discharge, Smears, Scrapings, Curettings.
3. Complete Blood Count.

Evidence of inflammatory changes doubtful history of venereal disease—

1. Blood Wassermann Test.
2. Gonococcus Fixation.

NERVOUS SYSTEM

Psychiatric examination is advisable in all cases showing nervous instability, evidence of emotional strain, and often in cases of high blood pressure, gastro-intestinal disturbance, headache, and other possible functional conditions; also where epilepsy is suspected.

There should also be complete neurological examination.

ENDOCRIN SYSTEM

Suspected thyroid disturbance—

1. Basal Metabolism Test.

Thirty-Fifth Annual Meeting

2. Sugar Tolerance Test.
3. Goetch Test.

Suspected pituitary change—

X-ray Examination of the Sella Turcica.

Examination of Fundus of Eye and determination of Visual Fields.

OSSEOUS SYSTEM

Evidence of focal infection, myositis, fibrosis, lumbago, etc.—

1. X-ray of Teeth.
2. X-ray of Sinuses.
3. Tonsil Culture.
4. Blood Wassermann Test.
5. Gonococcus Fixation.

Symptoms of pain or joint involvement—

1. X-ray of area involved.

THE SKIN

Skin tumors—

Biopsy.

Lesions—

Reference to skin specialist for microscopical and bacteriological examination.

Eczema—

Protein Sensitization Test.

MISCELLANEOUS HISTORY AND FINDINGS

History of residence in Tropics calls for—

1. Complete Blood Count.
2. Examination of feces for Parasites and ora.

History of malaria calls for—

Complete Blood Count.

Discussion—Health Examinations 93

Finding of sugar in urine, excessive overweight, or recent loss of weight with signs of diabetes, calls for—

Blood Sugar Test.

Family history of diabetes and obesity calls for—

1. Blood Sugar Test.
2. Sugar Tolerance if blood sugar is normal.

Summarizing my talk, I wish to emphasize the importance of not regarding the periodic examination service as constituting merely a *physical examination*. There are other important specialized phases to the complete service, if it is to measure up to a high scientific standard. It would be a pitiable miscarriage of science if any large effort is directed solely to the rendering of examinations and the separation of the sick from the well. There must be the ideal of constructive hygiene put into this work if it is to bring the best results either among policyholders or employees in industry or citizens at large.

I feel that we are just at the beginning of this work and that the next quarter century will see remarkable developments in the improvement and prolongation of human life. The life insurance business, like a great social giant awakening to action, has already made splendid contributions to public health but has not yet called into play its full reserves. There is no exaggeration in saying that with life insurance exerting its full power, it will have a more profound influence on human affairs than any other social agency. In this your association will find its splendid opportunity and I am sure will rise to it.

Dr. Ward—I am going to take the liberty of asking Dr. Geiringer if he will kindly tell the Association how they keep their records of these health examinations separate from their life records.

Dr. Geiringer—We make it a point in our health conservation service to say in all our literature that there is absolutely no connection between the life end and this health examina-

Thirty-Fifth Annual Meeting

tion branch of our service, and we follow through just as we advertise and just as we promise. The only connection is the determination of whether the individual who asks for the examination is entitled to that privilege.

Dr. Jenney—Would that be true if he applied for additional insurance?

Dr. Geiringer—Absolutely true. We never refer to the record.

Dr. Honsberger—I would like to add a word, Mr. President, as one of the Companies who has recently taken advantage of this—the Mutual Life of Canada. We were convinced that this would be a proper thing to do in the early part of this year, and I believe that about 10% of our policyholders have now received notice. We send our notices with the premium notice. About 10% have taken advantage up to this date—in less than a year's time. We assure our policyholders that it will have absolutely nothing to do with any future examinations, that there are no records kept nor given to other parties. Our policyholders are very well pleased with it. We have made no effort to force it on them, but merely send them a little slip with our premium notice, setting forth the advantages. It has proved a good advertisement for our agents, who are pleased, and our policyholders are invariably pleased. We find men who have been sitting in an office and taking no exercise, after taking the examination, playing golf, and men with high blood pressure consulting their physicians and taking their advice. We are well pleased with the results so far.

Dr. Knight—An important point brought out by Dr. Fisk is that 95% here in the city take that examination, and comparatively few outside. I am strongly convinced that the Medical Examiner for life insurance is not the man that ought to make these periodic examinations. He should know nothing about what those people show who come up for examination, and you never can keep that knowledge and

the use of that knowledge from your life insurance proposition if you are going to have the Medical Examiner in the field make those examinations. I think that is a very great mistake. I think the ordinary doctor in the field is neither trained nor equipped nor interested in this work. I do not believe that he is able to do it. It is worth while if done well and not worth a picayune if done badly. There is no use in throwing away effort. I think this is a question that should be seriously considered—the equipment of doctors especially for this kind of work, perhaps getting panels of local doctors who are eligible for it, perhaps securing them through hospitals and clinics, putting them through not just one agency but several agencies, and keeping this business absolutely independent of the life insurance business.

Dr. Steele—Along the line suggested by Dr. Knight—we are extending to all of our large policyholders the privilege if they are in the City of New York, of taking the Twenty-Dollar examination at the Home Office of the Life Extension Institute, and we are getting wonderful results from it. The large policyholders, nearly every one of them, are in New York at some time during the year, and in a small Company like ours, we are averaging about five of these Twenty-Dollar examinations a month at the present time, and we are getting remarkable results. In connection with that we run the same system that the Union Central does. The first year we had 10% of replies, the second, 12% and this year it will run probably from 15 to 18%.

Dr. McMahon—I am wondering if Dr. Toulmin can tell us whether an unduly large proportion of the people who apply for this examination are people who really think there is something wrong with them, or fear that there is.

Dr. Toulmin—I cannot answer that question because our experience has been so small. For two or three years we have had free examinations at our Home Office, but they are not nearly so extensive as those at the Life Extension Institute.

They have all kinds. I can answer the other question—there is absolutely no difficulty in keeping from your ordinary records these records of health examinations. They never appear in any place whatever in connection either with additional insurance or with restoration of lapsed policies. They are two wholly separate things.

In closing the discussion I wish to thank very sincerely those who have discussed the paper, both formally and informally, and to assure you that I have accomplished two of the things I hoped to accomplish. One was that you have shown me some very practical ways of continuing this work on a broader scale, and you have given me encouragement to carry on the work which I can confidentially tell you the Board of Directors has said may be carried on in as broad a way as the President and myself think is justified. I thank you all.

Dr. Ward—For many years we have been the recipients of most valuable information from the New York Life. Prior to this meeting they have given us two mortality studies, and today they are going to make another contribution, if I mistake not, fully as great in value as those which have preceded it. Dr. Rogers, will you present to the meeting at this time the subject before us?

MORTALITY STUDY OF IMPAIRED LIVES—NO. 3.

- (a) HEART MURMURS—MITRAL REGURGITATION.
- (b) FUNCTIONAL HEART MURMURS.

By DR. OSCAR H. ROGERS and ARTHUR HUNTER.

From an insurance standpoint, the most important type of heart murmurs is mitral regurgitation, not only because it occurs more frequently than any other in our routine work, but also because there are still wide differences among us in our treatment of them. Some of us do not accept these risks on any plan; some accept the best of them at standard rates,

while still others insure them only as substandard risks. In 1919, in a paper now out of print, we made a report of our experience with this type of heart murmurs and, with the hope that the experience of our company may be of service in helping all of us to discriminate more exactly between the various degrees of them, we have extended our investigation and have brought all our material down to the policy anniversary in 1923. With a view to broadening the basis of our investigation, we have included in our study cases with certain other impairments of a minor sort, such as malarial fever, if not recent or severe, a history of dyspepsia and the like. We are confident that these additions cannot have affected the mortality of the groups by as much as five points. The data are limited to men only, between 14% underweight and 24% overweight inclusive and to policies issued on a substandard basis. The study was made by policies. The expected deaths were calculated by the American Men Select Table, which fairly represents the Company's experience among standard lives on the issues of 1906 to 1922 inclusive, although our experience in the last few years of issue has been much lower than that table.

The material was studied in two groups:

- (1) Policies issued from 1896 to 1906 inclusive, usually with lien, on the Deferred Dividend Plan and in a special dividend class.
- (2) Policies issued from 1906 to 1922 inclusive, on the Annual Dividend Plan with an advance in age.

MITRAL REGURGITATION WITHOUT HYPERTROPHY

| | Actual Deaths | Expected Deaths on Standard Risks by A. M. Table | Ratio of Actual to Expected Deaths |
|--|---------------|--|------------------------------------|
| #1—Special Class (Issues of 1896-1906) | 324 | 174.8 | 185% |
| #2—Advance in Age (Issues of 1906-1922) | 481 | 252.6 | 190% |
| | 805 | 427.4 | 188% |

Thirty-Fifth Annual Meeting

As these groups were found to be in substantial accord, all of the material, which covers 13,219 policies, was treated as homogeneous, as follows:

MITRAL REGURGITATION WITHOUT HYPERTROPHY

| Policy Years | Actual Deaths | Expected Deaths on Standard Risks by A. M. Table | Ratio of Actual to Expected Deaths |
|-------------------------|---------------|--|------------------------------------|
| 1- 5 | 437 | 216.3 | 202% |
| 6-10 | 186 | 108.1 | 172 |
| 11-15 | 127 | 61.0 | 208 |
| 16 and over | 55 | 42.0 | 131 |
| All years | 805 | 427.4 | 188% |
| By Ages at Entry | | | |
| 15-24 | 141 | 64.7 | 218% |
| 25-39 | 425 | 210.9 | 202 |
| 40-49 | 168 | 97.5 | 172 |
| 50 and over | 71 | 54.3 | 130 |
| All ages | 805 | 427.4 | 188% |

It appears from the foregoing table that the relative mortality does not decrease until after fifteen years and, also, that it is higher at the younger than at the older ages of entry.

Of the 805 deaths, 339 (42%) were from heart disease. The number of deaths from heart disease in a corresponding group of standard lives would be less than 30. The death rate from pneumonia and from Bright's disease was twice the normal death rate from these causes.

In order to determine whether or not there has been an improvement in recent years, an investigation was made of the issues of 1919 to 1923 inclusive, carried to the 30th of June, 1924. It was thought to be inadvisable to take an earlier year of issue on account of the disturbance caused by the influenza epidemic in 1918-1919. The following were the results:

Rogers-Hunter—Impaired Lives 99

MITRAL REGURGITATION WITHOUT HYPERTROPHY

Issues of 1919-1923, Carried to the 30th of June, 1924.

| Actual Deaths | Expected Deaths on Standard Lives by A. M. Table | Ratio of Actual to Expected Deaths |
|---------------|--|------------------------------------|
| 145 | 84.2 | 172% |

In this connection it may be well to state that the mortality of our Company on its standard risks during 1919 to 1924 was considerably less than the A. M. Select Table.

We should like to remind our readers that in the paper published in 1919 the fact was brought out that the mortality was influenced by Build. As this point has already been covered, it was thought to be unnecessary to make any further study of that phase of the problem.

MITRAL REGURGITATION WITH HYPERTROPHY

The group of cases with hypertrophy consists of those in which the hypertrophy was sufficiently marked to require some additional rating for that condition. If it were so slight that the Medical Board did not think it was of moment, the case was included among those without hypertrophy. In the majority of cases the medical examiner would probably not report hypertrophy unless it were fairly well marked.

In the following table is given the experience on 3,633 cases of mitral regurgitation with hypertrophy for groups (1) and (2) combined, *i. e.*, for all policies issued from 1896 to 1922 inclusive:

| Policy Years | Actual Deaths | Expected Deaths on Standard Risks by A. M. Table | Ratio of Actual to Expected Deaths |
|--------------|---------------|--|------------------------------------|
| 1- 5 | 130 | 47.4 | 274% |
| 6-10 | 53 | 19.4 | 273 |
| 11-15 | 21 | 12.5 | 168 |
| 16 and over | 17 | 10.4 | 164 |
| All years | 221 | 89.7 | 246% |

**By Ages
at Entry**

| | | | |
|-------------|-----|------|------|
| 15-24 | 48 | 14.6 | 329% |
| 25-39 | 105 | 47.0 | 223 |
| 40-49 | 48 | 19.4 | 248 |
| 50 and over | 20 | 8.7 | 230 |
| All ages | 221 | 89.7 | 246 |

It seems probable from this study that the relative effect of the heart lesion decreases with the duration of the policy and that it is more serious at the younger than at the older ages at entry.

Of the 221 deaths, 101 (45%), were due to heart disease. The deaths from apoplexy and Bright's disease were distinctly above the normal.

DIFFERENTIATION OF SYSTOLIC APICAL MURMURS

Among experts in lesions of the heart, a systolic murmur at the apex is looked upon as functional or, at least, as innocent if there is no hypertrophy, no accentuation of the second sound over the pulmonic area, if the pulse rate and the blood pressure are normal and if the heart responds satisfactorily to exercise. With a view to determining whether such an opinion prevails in several of the larger companies which grant insurance at the regular rates of premium to carefully selected risks with systolic apical murmurs, we entered into correspondence with the medical directors of these companies, none of which issued any form of substandard policy. Two of these replies, which are representative of the others, are now given:

Company No. 1—"Where the murmur is not clearly conveyed, nor increased upon exercise, unaccompanied by other evidence of organic cardiac change, and especially when not heard constantly, it may be considered as non-organic in character, and acceptable at standard rates. Such murmurs may be heard chiefly in the recumbent posture and are usually

eliminated upon forced expiration, the heart sounds coming out clearly and the murmur disappearing, which is a fair indication that the cause is not due to actual organic disease of the mitral valve."

Company No. 2—"We consider any murmur, occurring in either part of the cardiac cycle system, indicates a valve lesion, no matter whether it is presystolic or diastolic or at the base or at the apex. We only accept murmurs systolic in time, heard either at the apex or base, and such murmur not transmitted to axilla; the apex in the normal area; no evidence of hypertrophy; no accentuation of second pulmonic tone; no abnormality of the pulse rate or rhythm and a blood pressure normal; no history of inflammatory rheumatism, tonsillitis or other acute infection and the examination must be made either at the Home Office or by an examiner of well-known ability and judgment."

It is evident from the replies to our questions that there is a serious effort on the part of companies which do not issue policies on substandard plans to select the very best of these risks with murmurs in the mitral area. There has also developed among the companies which do a substandard business, a tendency to differentiate more and more carefully in the treatment of mitral murmurs. It is no longer considered satisfactory to treat alike all mitral regurgitant murmurs without hypertrophy, nor may those with hypertrophy be accepted without regard to the history of the case or its freedom from other physical signs.

With the hope of shedding some additional light upon the subject, we have made, with the generous co-operation of our Medical Board, a thorough review of our material commencing with that of the middle of 1914, at which time we began to use a more efficient form of heart blank, on which our medical examiners were required to report the detailed results

102 Thirty-Fifth Annual Meeting

of their examination of the heart. The three groups into which the cases were divided may be summarized as follows:

A. Cases rated as mitral insufficiency in which the murmur, though constant, was slight and not transmitted; with no hypertrophy, no disturbance of rhythm or of function and with no history of rheumatism or other acute inflammatory ailment.

B. Cases rated as mitral regurgitation, constant, transmitted toward the axilla and, usually, heard at the angle of the scapula, without hypertrophy, without any complications and without any history of rheumatism or other acute inflammatory ailment.

C. Cases of undoubtedly mitral murmurs with no appreciable hypertrophy but which show some departure from the normal in the rate or rhythm of the pulse, in the blood pressure, in the accentuation of the second sound, or in which there was a history of rheumatism or other acute inflammatory ailment.

These murmurs may be designated for convenience, Group A, "innocent"; Group B, "average"; Group C, "unfavorable."

The differentiation was made by our Medical Board especially by Dr. E. H. Lines, Medical Director, by Dr. James H. North, Assistant Medical Director, and by Dr. Paul E. Tiemann, Assistant Medical Director, with a great deal of care in 4,565 cases issued from 1st July, 1914, to 31st December, 1918. Of these, 568 cases (12%) came under A; 2,744 cases (60%) under B; and 1,253 cases (28%) under C. In all cases a mitral murmur without hypertrophy was reported and there was no other impairment.

In order to broaden the material as much as possible the cases were studied as of the 30th of June, 1924, with the following results:

MITRAL REGURGITATION WITHOUT HYPERTROPHY

| | Actual Deaths | Expected Deaths on Standard Risks by A. M. Table | Ratio of Actual to Expected Deaths |
|--|---------------|--|------------------------------------|
| "Innocent" or Accidental Mitral Regurgitation— | (A) 18 | 17 | 106% |
| "Average" | (B) 134 | 85 | 158 |
| Mitral Regurgitation— "Unfavorable" | (C) 97 | 41 | 235 |
| Total | 249 | 143 | 174% |

Of the 18 deaths in Group "A" 3 (17%) were from heart disease; of the 134 deaths in Group "B" 47 (34%), and of the 97 deaths in Group "C" (44%) were from that disease.

Unfortunately, the amount of data in Group "A" is too small to enable us to draw any very satisfactory deductions from it. Whether groups of these so-called "innocent" murmurs rigidly selected, will show hereafter so favorable a mortality, cannot be determined from our material. It is not improbable, however, that as low a mortality may be obtained from a carefully selected group of such murmurs as among a corresponding group of functional heart murmurs.

With regard to Group "B," the results are in accordance with expectations.

The results in Group "C" show that the inclusion of apparently slightly unfavorable features has a more serious influence on the mortality than has generally been assumed. An analysis of the deaths from heart disease shows that in 55% of the cases there was a remote history of rheumatism (a history which would not call for a rating on account of the attack of rheumatism), of typhoid fever, of severe tonsillitis, or other inflammatory ailment.

In a previous paper we stated that in cases of mitral regurgitation a probable mortality ratio might be expected of as low as 150% in the best of them, and of as high as 225% or even 250% among the least desirable, depending upon the degree of hypertrophy, with a mortality in the neighborhood

of 170% in the average cases. In comparison with the A. M. Table our experience is 188%, but in recent years of issue it was 172%. It is probable that the insured with heart murmurs have not improved as a class as rapidly as have the insured generally. In drawing deductions from the ratios of mortality in special classes, we must have in mind always the low mortality of recent years, the higher mortality of the year prior to 1918 and the very high mortality during the influenza epidemic of 1918-1919. It does not, however, come properly within the scope of this paper to discuss the changing mortality on "standard" business and the difficulties which arise in measuring such mortality during the transition period.

FUNCTIONAL HEART MURMURS

Our experience with functional heart murmurs in recent years is not extensive under policies issued on a substandard basis, for the reason that we have been granting policies at the regular rate of premium at the younger ages, provided all conditions were satisfactory. Our experience, 3,459 policies, on risks treated as substandard for all years of issue from 1896 to 1922 is as follows:

FUNCTIONAL HEART MURMURS

| Policy Years | Actual Deaths | Expected Deaths on Standard Risks by A. M. Table | Ratio of Actual to Expected Deaths |
|----------------------|---------------|--|---------------------------------------|
| 1- 5 | 58 | 52.2 | 111% |
| 6-10 | 33 | 28.0 | 118 |
| 11 and over | 25 | 30.4 | 82 |
| All years | 116 | 110.6 | 105% |
| By Group Ages | | | |
| 15-24 | 29 | 29.1 | 100% |
| 25-39 | 53 | 56.3 | 94 |
| 40 and over | 34 | 25.2 | 135% |
| | 116 | 110.6 | 105% |

So far as it goes, the foregoing justifies the present treatment of our company, which is not to penalize any applicant

at the younger ages unless there should be other unfavorable features, but at the older ages to make about one-half the charge for mitral regurgitation.

Of the 116 deaths, 26 (22%) were due to heart disease—a very high proportion when the mortality for the whole group is considered. As 14 were among the group who entered under age 40 it seems probable that functional heart murmurs are of some importance even at the younger ages.

CONCLUSION

From the facts presented in this paper, we conclude that—

(1) The best cases of mitral regurgitant murmur, those which are not transmitted, will show a mortality but little above the normal. These are the border-line cases in which it is doubtful whether the murmur is actually organic or is simply "functional" in the mitral area.

(2) The average mitral regurgitation without hypertrophy, those which are transmitted, should not be accepted unless provision is made to cover a substantial extra mortality.

(3) Those mitral murmurs which present a history of rheumatism or some other acute infective process, will show a distinctly higher mortality than the average of mitral regurgitation.

(4) The cases of mitral regurgitation with hypertrophy show a distinctly higher mortality than the "average" cases without it, although a favorable type with moderate hypertrophy has probably about the same valuation as the "unfavorable" type without hypertrophy.

(5) Functional heart murmurs at the younger ages may be accepted at the regular rate of premium, if carefully selected, but a higher mortality than the normal should be provided for

106 Thirty-Fifth Annual Meeting

at the middle and older ages at entry. It is questionable whether persons engaged in hard physical labor should be accepted as standard risks if they have even a "functional" or "innocent" murmur.

Dr. Ward—Mr. Hunter, may we hear from you?

Mr. Hunter—I have nothing to add to what Dr. Rogers has said, except to point to the death losses as justification for our treatment. You will notice in Group B, which the doctor mentioned to you, the transmitted murmurs, that 34% of the deaths were from heart diseases; and in Group C, where there was a history of some inflammatory trouble, that 44% of the deaths were from heart disease. I know of nothing that justifies our treatment in the eyes of the agents than to be able to point to the death losses. If you tell them that your death losses among these heart murmurs are something like 15% greater than the deaths from heart disease would be among a corresponding group without the mitral regurgitation, you have got the agent where he cannot gainsay you.

Dr. Ward—It is a very great privilege today to have with us one who is recognized as an authority on this very difficult problem, and Dr. T. Stuart Hart, of New York City, whom many of you know by reputation, is going to speak to us at this time upon his interpretation of heart murmurs and cardiac arrhythmias. I feel that this is going to be a most profitable period for our Association. I take pleasure, gentlemen, in introducing to you Dr. T. Stuart Hart.

THE SIGNIFICANCE OF HEART MURMURS AND IRREGULARITIES.**DR. T. STUART HART.**

The honor which you have conferred upon me in inviting me to address this Association which has made such worthy contributions to scientific medicine is deeply appreciated. I have accepted your invitation, however, with considerable diffidence because I fully realize that I am unable to present to you the kind of statistical evidence which you rightly value so highly. My discussion must approach the subject from the clinical and pathological sides, my views must be drawn from the knowledge which has been accumulated by others and will be colored by a personal study of a considerable number of cases, but a number which is insignificant as compared with that which you are accustomed to treat statistically.

It should not be forgotten that the beginnings of selection on a physical basis were first suggested by the clinician. The recognition of the difference between an epithelioma and a wart was of value before they were studied statistically. Such obvious contributions to classification are no longer possible, yet I cannot believe we have yet reached the point where the view of the clinician is entirely without value in at least tentatively suggesting methods of classification and interpretation which may be of assistance in evaluating physical signs and in increasing the accuracy of the selection of insurance risks. Whether these views are sound or not, will be ultimately settled when sufficient material has been accumulated so that they may be tested statistically.

Today I shall confine my informal remarks to a presentation of my views in respect to those hearts which may safely be accepted at standard rates notwithstanding the presence of certain murmurs or irregularities. I shall not attempt to formulate my opinions in regard to the relative significance of the various types of organically damaged hearts. For a long

108 **Thirty-Fifth Annual Meeting**

time it has been my custom to make a definite prognosis for each individual with a defective heart who seeks my advice (this part of my opinion is not given to the patient except in exceptional cases, but is recorded in writing as a check for my own edification). I will only say that a comparison of my first impression and of the subsequent course have shown enough discrepancies to leave me in a very humble frame of mind in regard to my ability to formulate accurately a prognosis in every case of organic heart disease. I feel you will thank me for avoiding this phase especially since members of your Association have made such notable contributions to this subject.

It will perhaps limit our discussion if we make a broad statement in regard to heart conditions which, I believe, are not acceptable at standard rates.

- A. Congenital heart disease.
- B. Endocardial disease (Valvular disease).
- C. Myocardial disease.
- D. Pericardial disease which can be detected by physical signs.

HEART MURMURS

If we are to eliminate the above conditions, we may say that applicants presenting the following murmurs are not acceptable:

1. *A diastolic murmur.* This practically always means a mitral stenosis or an aortic insufficiency. To be sure there are a few exceptions to this general statement such as for example the Graham-Steel murmur of pulmonic origin, or rarely a diastolic murmur which appears to be due to no discoverable organic lesion (*Conner: L. A., Amer. Jour. Med. Sci., Dec., 1919., p. 776*), but these are so extremely infrequent that I feel they should not be considered as exceptions for the purpose we have in view.

2. *A presystolic murmur.* This means a mitral stenosis or rarely an aortic insufficiency (Flint's murmur).

3. *A systolic murmur associated with enlargement of the heart, or with a markedly accentuated second sound (either aortic or pulmonic) or with physical signs suggesting cardiac insufficiency.*

4. *A systolic murmur with maximum intensity in the second space to the right of the sternum.*

Applicants, presenting the following murmurs, should be acceptable at standard rates.

1. *Systolic murmurs heard with maximum intensity in the second and third spaces to the left of the sternum, provided there is no further evidence of cardiac impairment, such as abnormal pulsations, hypertrophy, cyanosis, clubbed fingers, other cardiac murmurs and cardiac irregularities (except sinus arrhythmia and extrasystoles).* These murmurs are extremely common in young people with flexible chests. They are more intense while holding the breath after expiration and may disappear on inspiration. They are sometimes associated with a systolic thrill which has no pathological significance.

2. *Systolic murmurs heard at or near the apex whether constant or inconstant provided:* There is no further evidence of cardiac disease, such as enlargement of the heart, abnormalities of the heart sounds, tachycardia or cardiac arrhythmia (except sinus arrhythmia and extrasystoles). A normal reaction to exercise. No history pointing to cardiac insufficiency. No evidence of disease of the kidneys. No evidence of disease of the arteries. No hypertension. No old history of acute infection such as rheumatism, chorea, recurrent sore throats and syphilis. No history of any kind of infection during the two years preceding the examination.

One of the very important factors in formulating an opinion on the significance of these murmurs is the *time element*. If

110 Thirty-Fifth Annual Meeting

a murmur which has the above characteristics has been known to exist for a number of years without any evidence of enlargement of the heart, it is hardly conceivable that the heart is seriously damaged. All organically diseased hearts (except a few cases of mitral stenosis which have other distinguishing features) show enlargement at a comparatively early stage. Krehl (*The Basis of Symptoms*, 1917, p. 17) says: "The increased work performed by the left ventricle leads to hypertrophy, a condition always present in mitral insufficiency."

I am inclined to think that any applicant, presenting a *slight systolic murmur*, no matter where located and which has been known to have been present for a period of at least five years, *provided* there is no history of acute infection, and no evidence of hypertrophy of the heart should be accepted.

When there is more than one impairment referable to the circulatory system such as impure or accentuated heart sounds, tachycardia, irregularities, moderate hypertension, etc., in addition to the murmur, acceptance should be guarded and only after a very thorough review of all the factors involved.

In my opinion (1) the *constancy* of a murmur is not a proper criterion upon which to judge its importance, it is true that murmurs, arising from organic disease are usually constant, on the other hand, we know that many murmurs are constant which are due to non-organic causes; (2) the *intensity* and *pitch* of murmurs may also lead us into error, the loudness of a murmur naturally impresses us with the gravity of the underlying cause, and yet we meet with many loud murmurs which cannot be explained by organic changes, a high pitched musical murmur may not infrequently be heard in hearts in which the post-mortem following death from causes other than circulatory failure shows no evidence of cardiac defect.

You will notice that the system of selection which I have presented is based upon physical signs and the history. I have intentionally avoided the question as to whether the classes

which are labeled "acceptable" have or have not organic lesions. I do not believe that it is possible in the present state of our knowledge to settle this question in each individual case and anyone who attempts to explain the cause of each one of these murmurs, will make many mistakes.

There are a number of facts which have lead to the views which we hold in regard to these groups. 1. Patients, belonging to these categories when followed for years almost without exception, fail to develop signs which indicate unquestioned organic lesions (I have a number of these under observation at the present time who have been followed over a period of 10 to 15 years). 2. Most of them stand up well under exposure and physical exertion for at least a few years, this has been made evident by the experience in our own and the British armies. 3. Autopsies on patients, dying from various causes, who have shown the stated abnormal physical signs usually show no discoverable heart lesion (occasionally an organic heart defect is found but this is very exceptional). 4. The stated restrictions eliminate those most liable to organic lesions, viz., the rheumatics and syphilitics. 5. These views are endorsed in part or as a whole by a number of physicians of large experience. Dr. Glentworth R. Bulter (*Jour. Amer. Med. Ass'n.*, Dec. 11, 1920, p. 1645) says, "It is, I believe, safe to say that a primary organic mitral insufficiency should, in no case, be diagnosed unless, in addition to the murmur, there is evidence of hypertrophy, and, preferably a definite history of rheumatism." Dr. Lewis A. Conner (*Amer. Jour. of Medical Sciences*, Dec., 1919, p. 779) expresses himself as follows: "I am inclined to believe that one will make fewest mistakes in the diagnosis of mitral regurgitation by adhering to the rule of never venturing a diagnosis on the presence of a murmur alone, no matter how characteristic it may seem to be," and further "It is believed that fully nine-tenths of all apical systolic murmurs belong to the class of accidental or functional murmurs."

In his report on behalf of the Medical Research Committee upon "Soldiers with Disordered Action of the Heart," Sir Thomas Lewis (Special Report Series No. 8, 1917) makes the following statements:

"Systolic murmurs at the base or apex indicate valvular lesions only exceptionally; there is no conformity of opinion as to the character or conduction indicating valvular lesion. The extent of valve damage which produces a systolic murmur alone is relatively slight; the disease is often limited to the valve, the heart muscle which is the essential part of the organ being wholly undamaged. Patients who are invalidated on the ground of systolic murmurs *alone* are subsequently found *when tested* to be fit for active service in nearly all instances. A large number of men who present such murmurs are known to have passed the most severe ordeals of active service without accident. If a group of patients who present no murmurs and a similar group in whom systolic murmurs exist, are tested in respect to their capacity for work or active service, no difference is to be found in the capacity of the two groups."

Since committing these views to writing, I have found a most excellent paper on this subject presented to your Association in 1917, by Dr. L. F. MacKenzie. His conclusions agree, with those at which I have arrived, in an extraordinary degree, although, of course, there are some minor points of difference. Dr. MacKenzie quotes the views of a number of clinicians of authority.

May I say that I do not like the term "functional murmur" as we use it at the present time. It seems to me to be an inaccurate use of this adjective and at times carries with it a wrong implication. All heart sounds and all heart murmurs, if we believe they are due to the activity of the heart, are functional. There are deviations from the normal sounds which are due to organic changes in the heart, others which are the result of conditions which are not organic. Would it not be more accurate and better convey our intended meaning if we should speak of a murmur as due to an organic or

a non-organic change as the case may be? If objection is made to using the term "non-organic" for these murmurs the cause of which is unknown the term "accidental murmur" would seem more satisfactory than the term "functional."

One word in regard to "functional tests" of the capacity of the heart. At one time it seemed as if a determination of the "vital capacity" would be of considerable value in settling this question. My own experience with this method has been disappointing, it is of distinct value in following the fluctuating capacity of the individual but it is of very little use in making an early diagnosis of muscular insufficiency. By the time that the "vital capacity" is diminished to the point that it is of diagnostic importance, other more obvious symptoms are always in evidence. Personally, I feel I derive the greatest amount of information from a simple exercise test, *i. e.*, running, hopping or stairclimbing. I do not believe in using a fixed amount of exercise for all individuals whether measured by the number of steps or foot-pounds, since it is obvious that 100 hops for example will put a very different tax on the trained athlete and the man of sedentary habits. Exercise should be adapted to the individual so that it will be severe enough to produce a slight degree of dyspnoea. The appearance of the applicant during this test with a determination of the heart rate before, immediately after exercise and after two minutes rest will give one as much information as can be gained by employing the more elaborate methods. (I regret to say that I have had no personal experience with the functional test recently presented before this Association by Dr. Frost).

There is another method of estimating the functional capacity of the heart which, in my experience, is of more value than any other, that is the information secured from a carefully taken history. If, in the ordinary activities of life such as stair climbing, running for trains, carrying hand baggage

114 Thirty-Fifth Annual Meeting

and the like a man develops no symptoms which suggest cardiac insufficiency one may be pretty sure that the heart muscle is efficient. To secure this information, the history must be taken skillfully and leading questions are to be avoided, one must take time for this process and the propounding of a series of direct questions will not always elicit the desired information. The information is often best obtained by an indirect approach giving one an opportunity of studying the psychological reactions of the subject. I fully realize that this method is likely to be of less value in applicants for insurance than in the ordinary practice of medicine, since a correct history is much more difficult to secure in the first group.

CARDIAC ARRHYTHMIAS

As far as I know up to the present time irregularities of the heart have been studied statistically only as one large and undifferentiated group. It is my belief that the sooner these statistics are discarded and forgotten the sooner will we be in a position to make a new start on a more rational basis. It would not be of less value to estimate the risk of infections by including in one group mumps, typhoid fever and pneumonia than has been the custom to evaluate the significance of cardiac arrhythmias as one undifferentiated mass.

Applicants, presenting the following types of irregularity, are not acceptable at standard rates.

1. *Heart block* (including delayed conduction, partial block and complete block).
2. *Auricular fibrillation.*
3. *Auricular flutter.*
4. *Alternation.*

Prolonged clinical observation has shown that each one of these types of arrhythmia is either associated with, or there later develops a condition of circulatory insufficiency.

Painstaking pathological studies have shown that with few exceptions the underlying cause is a diseased myocardium. The exceptions are so few that in most instances, it is believed that the failure to discover damaged muscle is due to the inadequacy of our present methods of examination rather than to the absence of an organic lesion.

When we come to consider the (5) *paroxysmal tachycardias*, our grounds of rejection are less secure, but I do not believe that even with an otherwise sound heart we are at present justified to consider them standard risks. Some of these are paroxysmal fibrillators and subsequently become continuous fibrillators. In other words, the mechanism is a temporary flutter. I believe, although this has not as yet been proved, that there is a real myocardial change in these cases. Others have these paroxysmal attacks from childhood to advanced years without developing cardiac insufficiency and without any deviation from health except the temporary discomfort. This latter group is not a large one and in the light of our present knowledge cannot safely be taken at standard rates.

Eliminating the above groups, there are left for our consideration two types of arrhythmia.

6. *Sinus arrhythmia*.

7. *Extrasystoles* (premature contractions).

Sinus arrhythmia should not be considered an impairment. It is merely an exaggerated phase of a physiological condition. It is extremely common in children and young people. Even in adults, minute measurements made by instrumental methods will reveal a slight variation in the length of successive heart cycles. These irregularities are caused (by slight variations in the control exerted on the heart by the pneumogastric and accelerator nerves. In the most extreme types (unaccompanied by other abnormalities) I have never met with a case that showed evidence of cardiac insufficiency, nor have I ever seen one so reported in the literature.

116 Thirty-Fifth Annual Meeting

Extrasystoles are extremely common. According to Sir James MacKenzie, there are probably very few persons who reach the age of 50, who at one time or another have not had at least a few of these abnormal contractions. The exact cause is not definitely known, but some toxic agent such as tobacco and nervous influences such as arise under various emotional conditions frequently act as exciting causes. My personal belief is that there is an underlying slight myocardial defect although in the majority of instances, no such lesion has been demonstrated by the pathologist. In most cases, this muscle damage is so slight that it should not be regarded as an impairment. When these abnormal beats are infrequent and transitory and all arise from the same point in the myocardium (which is usually the case) the applicant should be accepted as a standard risk. Extrasystoles which are frequent and persistent, and those which arise from several points of origin should make one suspicious of more serious damage to the myocardium and if they cannot be controlled by appropriate treatment they render the applicant unacceptable at standard rates. Abnormal beats of this character arising from more than one focus in the heart muscle can only be differentiated by an electrocardiographic examination.

A statement of what types of arrhythmia are acceptable and what are not seems to me rather a simple matter but the technique of selection to be employed by the Life Insurance Company appears to me to offer considerable difficulty. The company must depend to a considerable degree on the reports sent in by examiners all over the country many of whom have had very little inclination or opportunity to perfect their knowledge of the differentiation of the several types of arrhythmia. Until recent years the practical importance of this subject was known only to the few. Today the average practitioner will throw up his hands when one begins to talk about "extrasystoles" and "flutter." It is often amazing to me how

frequently one will meet with a practitioner of standing who will glibly use the terms "heart block" and "auricular fibrillation" as if they were interchangeable and evidently without the most remote idea as to their significance. On the other hand, the recent hospital graduate is usually quite proficient in distinguishing the various types of arrhythmia. I feel that for the present every applicant reported as having an irregular heart should be required to submit to an examination by some one who is known by the Insurance Company to have had experience in the diagnosis of these cases. The diagnosis is not difficult to one who has had a reasonable amount of study and experience along these lines. I think it may be safely stated that 95 per cent of all arrhythmias can be correctly differentiated by the ordinary methods of examination without recourse to the help of instruments of precision.

* THE ELECTROCARDIOGRAPH

If I were asked what is the most important function which the general use of the electrocardiograph has performed, I should say the education of those who make use of it. It stimulates an interest in the study of heart conditions and acts as a check on findings made by other methods. A man who knows that his diagnosis is to be reviewed by a method which within its field is absolutely accurate, will exercise considerable care in committing himself and will have the further advantage of a means of correcting the errors which will occasionally occur.

Both the polygraph and the electrocardiograph are useful instruments in studying heart conditions. They, of course, record different phenomena, the polygram is a time pressure curve, the electrocardiogram is a graphic portrayal of the variations of electrical potential during muscular activity. If one is to use only one of these methods, the electrocardiogram will be found the more useful. It is much easier to obtain

a satisfactory curve with the electrocardiograph than with the polygraph. The electrical method records a wider range of abnormalities and the curves are more quickly read.

The electrocardiogram is the most accurate method which we have of analyzing all types of cardiac irregularities except "alternation." In addition to this, it discloses certain myocardial defects which can be detected in no other way, for example the lesions known as "bundle branch block," "arborization block," and certain types of extrasystoles arising from multiple foci. Most of these lesions belong to rather advanced conditions of muscular damage and probably very few would be encountered in life insurance applicants, excepting those belonging to the substandard groups.

I thought it might be of interest to you to look over two or three electrocardiograms of the types which have been referred to and, therefore, will pass them around for your inspection. From these records the ordinary (harmless) type of extrasystole is easily picked out, when the extrasystoles are frequent and arise from more than one point the records are equally obvious.

Another curve shows a rather uncommon condition, a blocking of the right branch of the bundle of His. This was secured from a lady of 50 years of age, who had no symptoms which could be definitely ascribed to the heart, and no abnormal physical signs. The heart was normal in size, position, rate and rhythm, the sounds were of good quality, there were no murmurs. This serious condition could have been discovered only by means of electrocardiographic records. She died of heart failure within 3 years of the discovery of the lesion.

That this is a grave condition is brought out by a study of this group which I have recently made: During the past 6 years I have seen 25 of these cases and have been able to trace the outcome in 21, of these only 3 are living at the

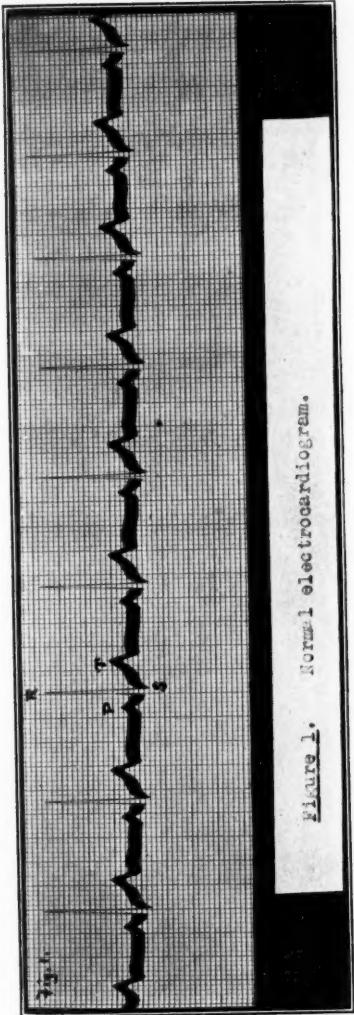


Figure 1. Normal electrocardiogram.

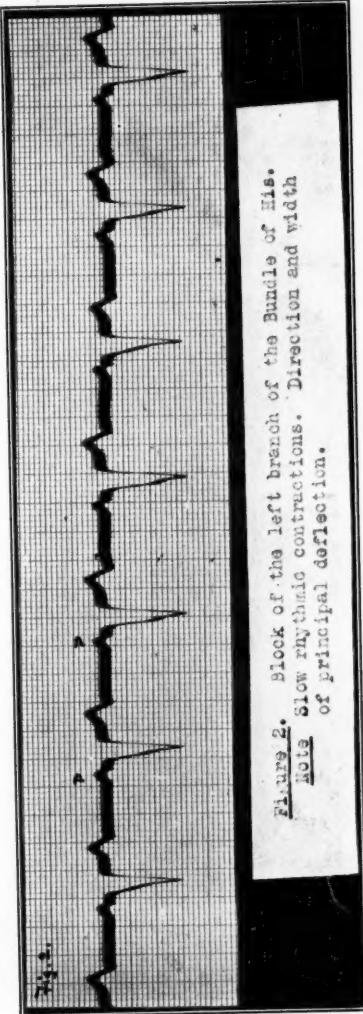


Figure 2. Block of the left branch of the Bundle of His.
Note Slow rhythmic contractions. Direction and width
of principal deflection.

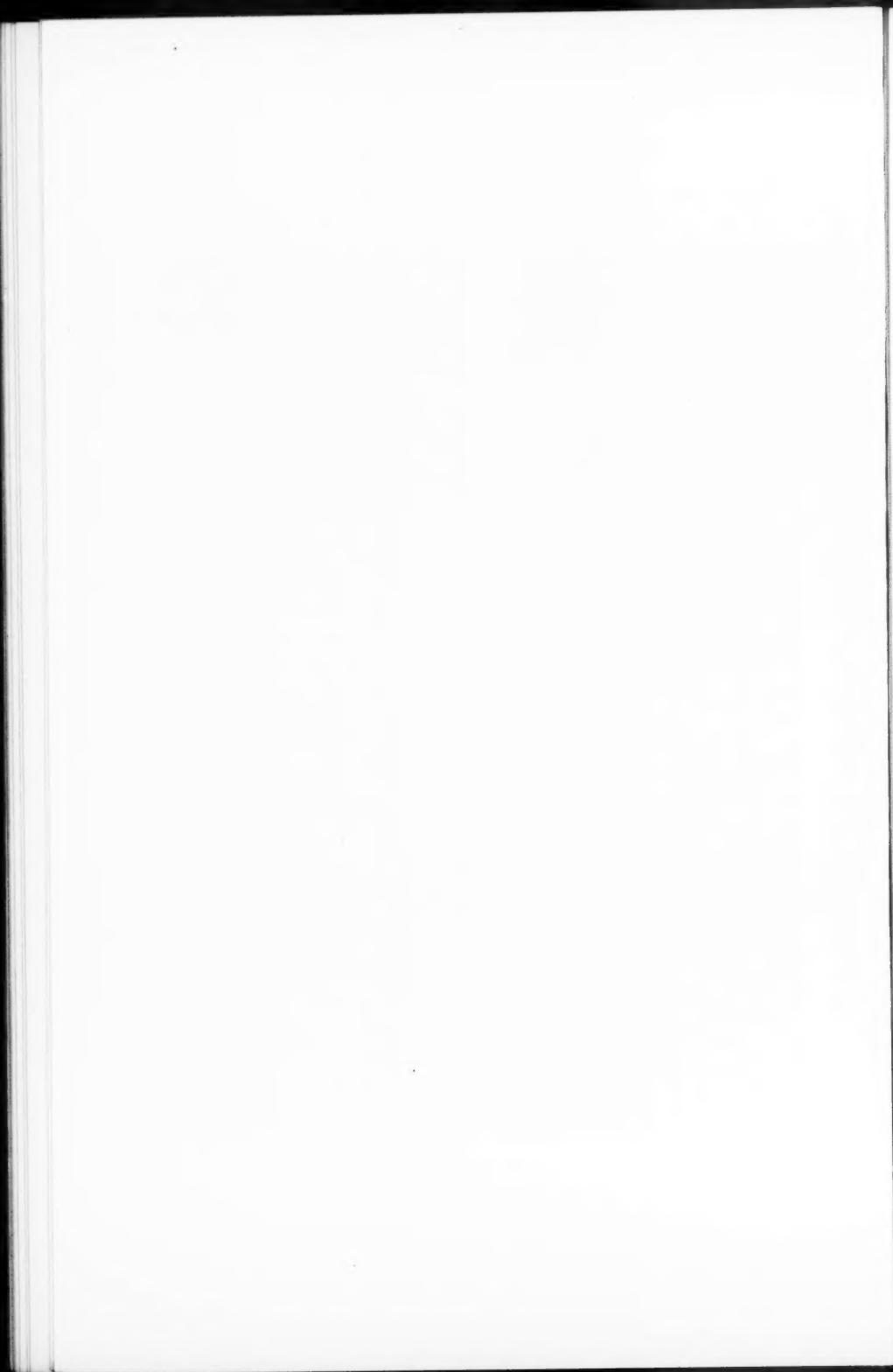


Fig. 3

Lead III

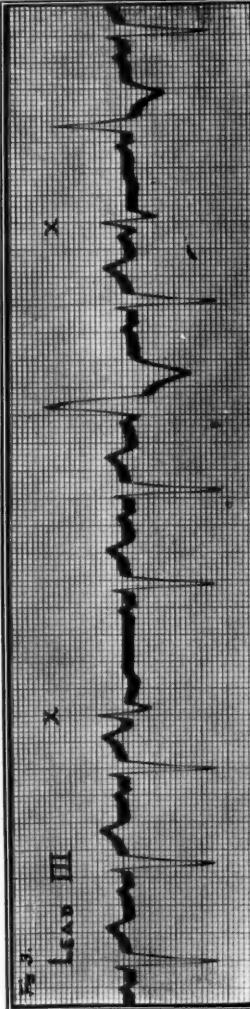


Figure 3. Extrasystoles - frequent and arising from several points in the ventricle.

Fig. 4

Lead II

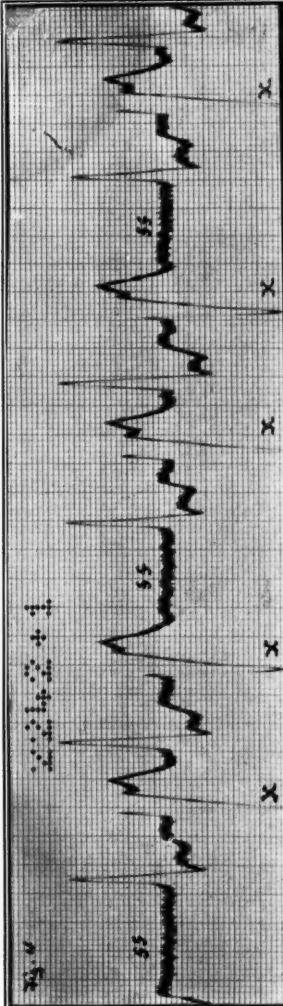


Figure 4. Extrasystoles - frequent and arising from several points in the ventricle, complicating atrial fibrillation.

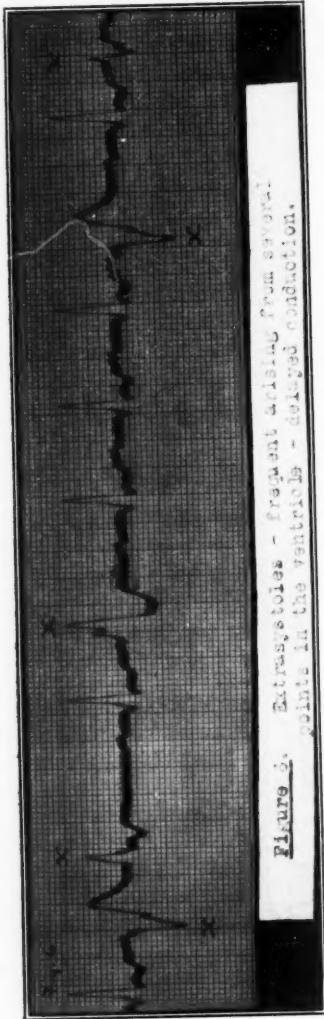


Figure 2. ECG tracings - Frequent atrial fibrillation points in the ventricle - disturbed conduction.

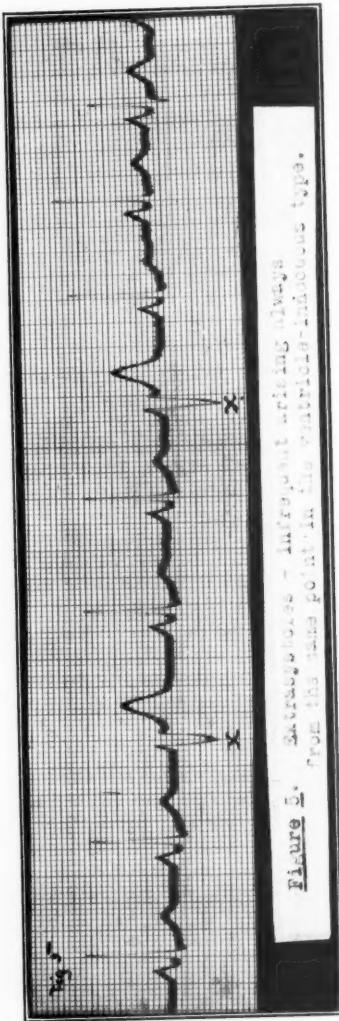
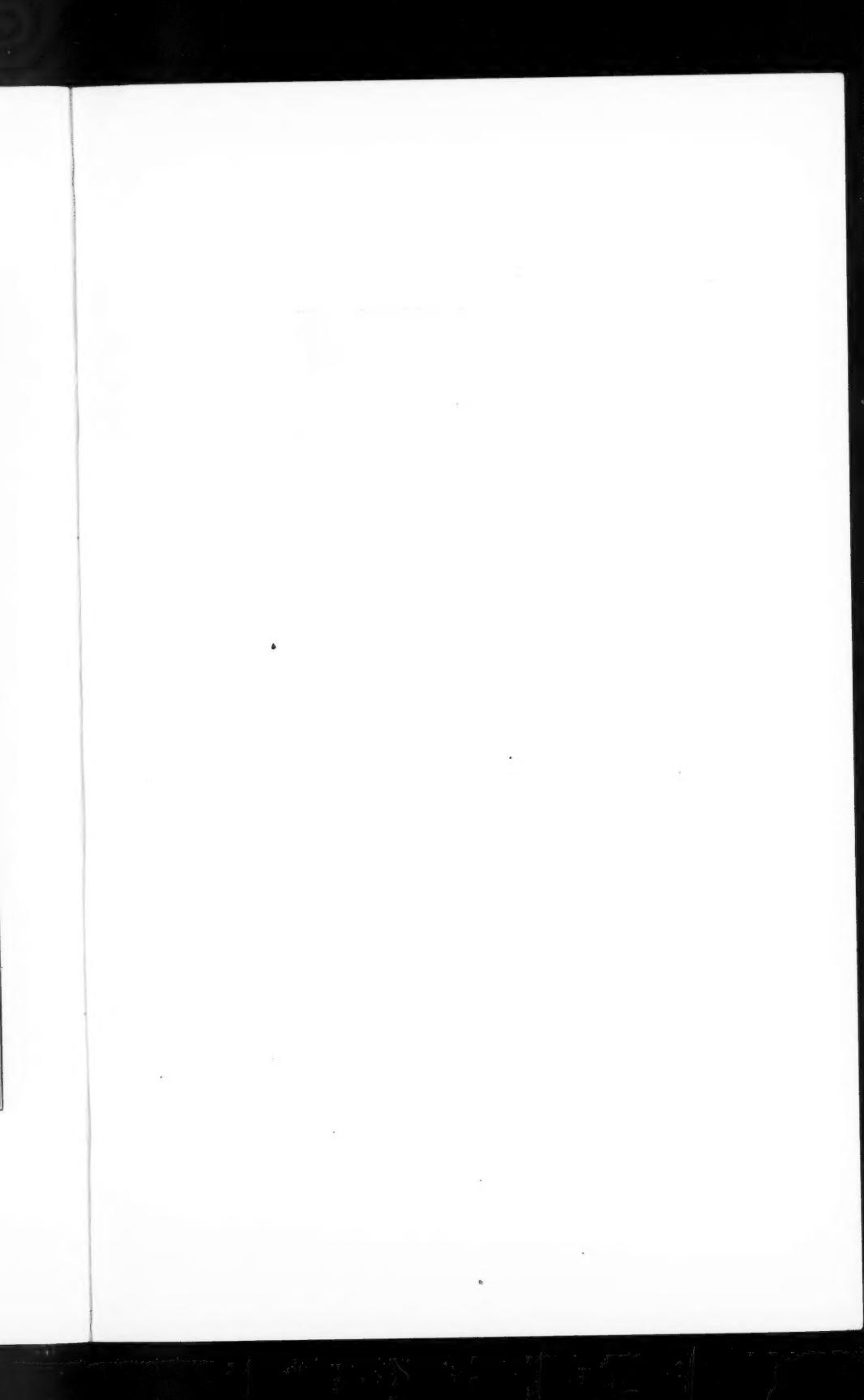


Figure 3. ECG tracings - Infrequent ventricular fibrillation points in the ventricle - disturbed conduction.



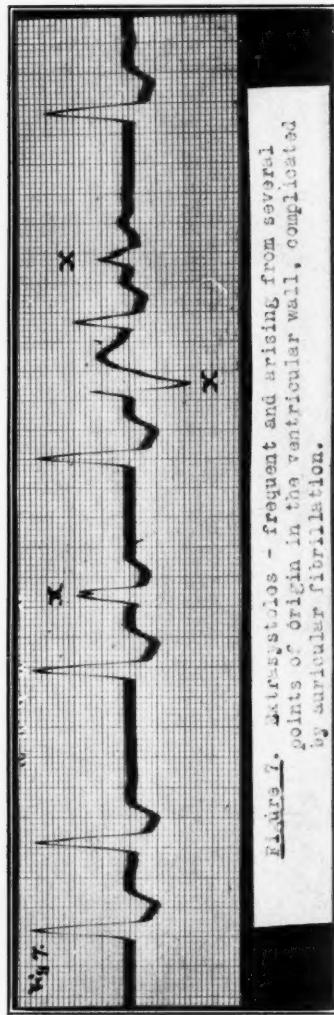


FIGURE 7. PVCs - frequent and arising from several points of origin in the ventricular wall, complicated by auricular fibrillation.

present time with observations respectively covering periods of 4 years, 14 months and 10 months. 18 are dead, most of them died within 6 months after the electrocardiographic evidence was obtained.

The electrocardiogram also furnishes us with information as to the relative muscle mass of the two ventricles, this is at times of considerable value in estimating the extent of some obscure injuries to the heart.

Those unaccustomed to reading electrocardiograms are apt to be appalled by the forbidding appearance of these curves, which seem to be as obscure and as difficult of interpretation as a group of Egyptian hieroglyphics. I can assure you that this is a most erroneous conception, and that a few hours' systematic study will enable any physician to read intelligently the simpler records and it is only a matter of a somewhat more extended practice to decipher all that anyone knows about the most complicated. This statement is based upon a considerable experience in introducing medical students to this aid in diagnosis.

X-RAY

I wish also to go on record in urging the more frequent use of the X-ray for a study of heart conditions. Here again I feel that this method of examination is chiefly of value as a means of education, serving not to replace but to improve and check the methods usually employed for estimating the size and position of the heart. Either the fluoroscope or photographic plates may be used for this purpose.

I have tried to present to you some of my views in regard to hearts presenting certain abnormal physical signs. In order to test the validity of these opinions, it will be necessary to accumulate cases enough to test them statistically. No one individual can do this, but an organization such as yours can do it if it seems to them worth while. To my mind even such

120 Thirty-Fifth Annual Meeting

statistics will fail of their object unless the classes studied are rigidly grouped. If you should undertake such a research, I should commend to you the more frequent use of the electrocardiograph and the X-ray. This would tend to add further accuracy, would be of distinct educational value and would serve to protect the interests of both the applicants and the companies. It is not likely that these laboratory instruments can be used in the great mass of cases, but at least a beginning could be made by securing the information which they afford in certain doubtful conditions and cases in which the amounts at stake are large.

Dr. Ward—Has Dr. Root something to say to us on this subject?

Dr. Root—Mr. Chairman—I don't know when I have been as interested as I have today to hear a paper that reverts once more to the good old days of clinical medicine, and reverses the actuarial point of view of life insurance!

While of course in estimating the value of a life from the group standpoint, the actuarial figures are absolutely essential, nor can a life insurance enterprise be carried on without figuring the mortality of the group, there still remains the personal equation and the individual risk who as a member of that group is a factor.

The paper we have just heard is to my mind a most interesting and valuable contribution to the study of physical diagnosis with regard to heart lesions, and I could only wish that the doctor had elaborated much more in detail and perhaps gone further. Since the days of Sir James Mackenzie's first book, a matter now of fifteen years, I think, and the more recent studies of our friends in clinical medicine, there has been a great change in opinion regarding the value of heart murmurs. I remember a few years ago, Dr. Christian, before the Massachusetts Medical Association, expressed views almost identical with those that Dr. Hart has set forth, and I remember most distinctly in the discussion that these views were re-

garded as having a revolutionary trend. Time has justified Dr. Christian's views, and will undoubtedly justify Dr. Hart's views.

I should like to ask in this connection, speaking more especially of the differentiation of the apical murmurs, whether the Doctor considers the age factor as important. I mention that because our earlier life insurance investigations show very distinctly that age had a strong influence on the mortality of large groups. You will find among the old records printed in the Mutual Life Office, twenty-five or thirty years ago, that the murmurs among young people, up to say thirty-five, show an excellent mortality, whereas the irregularities of middle life and later showed a very much higher mortality, and I should like to inquire regarding the age factor; and also to ask whether the difference in position or attitude enters into the question in differentiating the various murmurs one from another. It has been a very illuminating paper, and I thank you very much for it.

Dr. Ward—Dr. Hart, will you answer that question as to the influence of age on these murmurs?

Dr. Hart—Of course there are so many exceptions, but I do think that a systolic murmur is regarded much more seriously with advancing years, and the absence of hypertrophy has to be taken into consideration, because we have a good many cases from 45 to 50 years of age, with no hypertrophy, and a systolic murmur.

As regards examining the patient in different positions, I cannot find anything that helps me in making a diagnosis of difference in murmur except corroborative signs. I cannot find that position or attitude helps.

Dr. Root—Would you venture any explanation as to the cause of these murmurs?

Dr. Hart—I do not think I have anything new to offer along that line. There are a great many explanations, many of them more or less hypothetical.

122 Thirty-Fifth Annual Meeting

Dr. Ward—Has Dr. Sykes anything to tell us on this subject?

Dr. Sykes—It would be impossible to add anything to these two excellent papers that have been presented, but the thought occurred to me when Dr. Ward asked me to say a few words that the new classification that the M. I. B. Committee has recommended is going to be of marked assistance to us in Home Office work, in interpreting, particularly at the younger ages, the different types of heart murmurs. Last week, we received an application which the examiner diagnosed as mitral insufficiency. We sent him a heart blank, and it came back and showed that the man had a presystolic murmur.

Another illustration—while serving at Fort Grant I was asked to address the medical officers on heart murmurs, and I thought it an excellent opportunity to find out from a group of one hundred physicians from various parts of the country as to how they would interpret heart murmurs—and this goes back to the question of the M. I. B. classification—I looked around, and found three cases that I thought typical, and I demonstrated them. One case was a local systolic murmur at the second interspace transmitted a short distance; another case was a systolic murmur at the apex transmitted down to the axilla and posteriorly; another was a case of cardio-respiratory murmur. And invariably the diagnosis was made of mitral insufficiency. It was most interesting to hear the comments of the various medical men, after demonstrating by forced expiration or inspiration how the cardio-respiratory murmur disappeared, and then drawing on the chest the location of the murmur and the transmission. It seems to me that there is a big field of education along this very line, and I think that with this new classification we will have to do it, because in the past our information has been what we got in the diagnosis—we have been pretty well satisfied to go that far, forgetting the symptoms.

Dr. Ward—Dr. Baker, have you a word for us?

Dr. Baker—I believe, Mr. President, that in dealing with this question, we should look at the practical side of it. If we had examiners throughout the field like Dr. Hart, we could accept a great many of these heart murmurs, but where the report comes in from the ordinary country doctor that there is a mitral systolic lesion or a murmur at the apex, I think it deserves considerable more attention, and in my opinion we must always look at this thing from the point of view of the average examiner in the country.

Dr. Ward—We still have a little time for further discussion. Possibly some of you gentlemen would like to ask Dr. Hart some questions. The discussion is thrown open.

Dr. McCrudden—Mr. President and Members of the Association—As I am one of the newer members I cannot contribute anything to the real life insurance aspect of this question, but I have been interested in hearts, and I have had two experiences that I thought might expand a little bit Dr. Hart's statement early in his paper that the clinicians first have to define the groups they are dealing with before the life insurance investigators can determine the mortality. A few years ago I happened to be connected with one of the hospitals that took care of veterans, and there were ninety-five doctors in the district who sent cases of all kinds to the hospital. A great many cases of alleged heart disease passed through my hands, sent by these ninety-five men, who were considered competent and who had been appointed as physicians by the government. I got so many cases that were considered serious heart disease, serious enough to be sent to the hospital, in which there was no evidence of heart disease whatever. We took a group of one hundred, just to get some definite statistics, and to find out how many cases of heart disease there really were in that group and why they were sent to the hospital for heart disease. In the group that I studied, over a period of fifty days, one hundred cases of alleged heart disease, there were 45 cases of effort syndrome pure and

124 Thirty-Fifth Annual Meeting

simple, with no evidence of any heart disease whatever, and there were 34 cases of heart disease. These 45 cases of effort syndrome were sent chiefly because of sinus respiratory arrhythmia, extra systoles, mitral murmurs, and certain symptoms, dizziness, attacks of fainting, shortness of breath, pain around the heart. That is one experience, and as the time is so short I won't develop that any further.

The second experience was this: This spring for the New England Mutual I made a visit through the west. While I was there some of the difficult cases were referred to me of all kinds in order to see if we could reverse our decision. I think about half of all the cases I saw were cases of effort syndrome that had been declined on the basis of what some of the doctors had diagnosed as possible heart disease. Now, what is the life insurance aspect of effort syndrome? It is very common. I don't know what proportion of young people have it, but it is certainly very large. There are a great many young men who have heart murmurs of just the type described by Dr. Hart, heard in the second and third interspace to the left of the sternum, heard best when the applicant holds his breath in complete expiration and accentuated by exercise. A great many of these men have extra systoles which disappear when taking exercise. A great many have sinus respiratory arrhythmia murmurs that are so marked that you have to listen quite a while to make absolutely certain that it is not auricular fibrillation. A great many of these men have pain around the heart, shortness of breath, and they exhibit obvious nervous symptoms. They have lively knee-jerks, their fingers will tremble when they hold them out. Very often they will have a blood pressure of 150 and I have seen 160 in a man who I am sure ought to be an acceptable life insurance risk. Now I don't know that these are acceptable life insurance risks. As clinicians we do tell these men that this condition is very common in young people, it is nothing that they need apprehend will be serious to them,

that it will wear off. We do that because, I think, the experts in heart disease, like Mackenzie have done it. But those cases have not been followed long enough to know what the mortality is. However, effort should be made to define those cases, so that our examiners can recommend them and we can determine the mortality on them. It is a very large group and a very important life insurance problem.

Dr. Ward—Dr. Mackenzie—Have you a word to say on this subject?

Dr. Mackenzie—I have listened with a great deal of interest to Dr. Hart's paper. In it he made a statement that I might say rather surprised me, for I have been acting in such a contrary manner upon these cases. He seemed to think that a systolic murmur could be taken at standard rates even with an extra systole. Am I right in that, Dr. Hart?

Dr. Hart—Yes, quite right.

Dr. Mackenzie—It would seem to me that while an extra systole did not necessarily mean a pathological condition of the heart muscle, that it is at least on the border line between the physiological and the pathological, and that if we have such distinct evidence of some irritation of the myocardium as is given by an extra systole, that we have got to go pretty slowly in taking such a case, if we also find a heart murmur, because whether a heart murmur is functional or organic, I think we have got to recognize the fact that it is an evidence of increased strain on the heart muscle, and that if we have with this heart murmur an extra systole, we have distinct evidence that there is irritation of that heart muscle with the increased strain. For that reason I have felt that I would be taking a good many chances if I passed on such cases, at least at standard rates, or even at a moderately increased rate.

Now, there is a question that has exercised me a great deal in connection with my reading in heart work. I have read everything that Dr. Rogers and Mr. Hunter have given to this

Association, and read it with a great deal of profit and interest. There has been in my mind a question all the time, how far they have gone in demanding a functional test, what functional test they have used, whether in all these cases a blood pressure reading was taken, whether the blood pressure reading has also been used as a functional test, and if complete urinalysis at the Home Office has been made in these cases. I noticed in the last paper that the mortality given by them for mitral regurgitation, in the past five years, I think, seemed to be better than it had been previously, and I wondered if the improvement in mortality could in any way be ascribed to, we might perhaps say, a more rigid selection of the cases through increasing the requirements regarding the functional test, the blood pressure and the urinalysis.

Now, I would like to know what Dr. Hart thinks from an insurance standpoint about accepting these cases, particularly around 35 to 40 years of age. It may be all right from a clinical standpoint, but from an insurance standpoint, would he want us to take cases at these ages that have an extra systole and a systolic murmur at the apex?

Dr. Hart—I should be inclined to take them. I may be mistaken about these things, but that is my view at the present. I think they are both apparently innocuous.

Dr. Ward—Gentlemen, we are going to have another period which will be just as profitable as the last one has been. It is a great privilege to have with us at this time one whose name is known to us all, and who is, I am sure, going to give us a very great contribution, for there is no problem that is greater or that presents greater difficulties to the Medical Director than these cases of suspected tuberculosis. I take great pleasure in introducing to you Dr. James Alexander Miller, who will speak to us on the Relative Value of the Various Factors in the Diagnosis of Suspected Tuberculosis. Dr. Miller will illustrate his lecture with stereoptican slides.

**RELATIVE VALUE OF THE VARIOUS FACTORS IN
THE DIAGNOSIS OF SUSPECTED
TUBERCULOSIS.****DR. JAMES ALEXANDER MILLER.**

It is a very great privilege to come and speak to this body of men, because it gives me an opportunity to say things which I have thought many times, but have never had an opportunity to say to you collectively. In other words, there are some things which an outsider sees which may be of some interest and value to those who are responsible for the medical policies of the life insurance business. On the other hand, it is a privilege, because I really expect to learn something from the point of view of the life insurance Medical Director about some of these problems in tuberculosis which, as your President has said, puzzle us all.

I cannot hope to really contribute very much to the collective knowledge here represented, because I do not know of any group of men that really know better what happens to cases of tuberculosis than life insurance Medical Directors. They have the habit of analyzing statistics, and the opportunity of studying large numbers of cases which gives them a clinical experience of this type which cannot be vouchsafed to anyone, no matter how large his experience, in private practice.

Now, in this question of tuberculosis there are of course problems which are particularly important from the stand-point of life insurance examinations. They are, however, similar, although different, from those with which we are all familiar. I shall keep in mind the particular point of view in which you are interested, but I shall at the same time try to present to you some of the things regarding tuberculosis which it seems to me are important for us all to understand, and perhaps by presenting it in this way, my own point of

128 Thirty-Fifth Annual Meeting

view may be broadened, and at the same time we may be able to arrive at conclusions which will be of mutual advantage to us all.

I have been accustomed to think of tuberculosis from the standpoint of diagnosis, and that is what I shall first speak of, and it is fundamentally composed of three concepts which it is important for students of the subject always to bear in mind, and which I think frequently are not.

The first is, that tuberculosis in itself is a relapsing disease, manifesting itself, not as a rule by any continuous set of symptoms or physical signs, but by interrupted periods, by exacerbations with symptoms, and with greater or lesser periods of quiescence. This is important, I think, from the standpoint of diagnosis, because it comes into the question of understanding what relationship the cold of today may have to the grippe of last year, with the pleurisy with effusion of five years ago, with the ischio-rectal abscess of eight or ten years ago, with the malaria of five or six years ago, how they all when properly interpreted spell one and the same thing, and linked together make a definite clinical picture, although the patient comes to you and says that he was well until a short time ago, caught cold, &c. I think is extremely important from the standpoint of the clinician not to get a history which is often very misleading, and perhaps in that way puts us off the track, while for you life insurance men, paying as you must of necessity, less attention to the history than we do, are perhaps not so apt to fall into this particular error. But I think it is extremely difficult for the ordinary man who does not see tuberculosis in its course to understand that the course of tuberculosis is just this interrupted one, and if we are going to understand and properly interpret the particular findings of today, we must interpret properly these exacerbations which have been labelled with other names in the minds of the patients and of their practitioners at the time, and we must call them what they really are, that is, different mani-

festations of this same disease. This is not only important from the standpoint of the understanding of the history and the diagnosis, but it is extremely important from the standpoint of the course of the disease while under treatment, and also, and perhaps most important of all, for a group like this, from the standpoint of prognosis.

The second fundamental concept which I have of the disease is, that it has in general two sets of manifestations (and I am speaking now of pulmonary tuberculosis), the focal and the constitutional. I find that practitioners of medicine as a rule pay a good deal more attention to the physical signs than they do to the constitutional symptoms. I believe that is because we begin as medical students seeing tuberculosis taught as exemplifying various sorts of physical signs. Every one of us were taught physical diagnosis of the lungs from cases of pulmonary tuberculosis. Consequently from the time we were third year medical students we thought of tuberculosis in the sense of interpretation of certain physical signs, when, as a matter of fact, we find when we come to know the disease better that the interpretation depends less on the physical signs than on the constitutional reaction. I think it is very difficult to understand unless you are seeing a good deal of tuberculosis, that it isn't so much the extent or the type of the signs or the rales you hear in the chest, as it is how that particular patient reacts to the disease which is important.

The third concept I think is very important from the standpoint of life insurance men, and particularly now, when so many groups are being examined who are apparently well—people who are not coming to a physician because they are sick, but are coming to be overhauled, and that third concept is the fact that a tuberculosis infection leaving behind perhaps its scar, is not in itself a disease.

The reason that I emphasize that point is that we know that most adults—between 90 and 95%—are infected, and

we know that most of us have never thought of ourselves as having tuberculosis. More and more, as we examine people, we know that if we go into it carefully enough we will find abnormalities in the chest which need interpretation, and if you belong to the school of medicine in which you have been taught that physical signs are the important thing in tuberculosis, and you listen to the chest of a man who is perfectly well, and you hear a few crackling rales above the clavicle, you immediately associate it in your mind as apical tuberculosis, which means to most people a person who is seriously ill and incapacitated, and from the standpoint of life insurance a bad risk, to be discarded. Many such cases are consequently put into the discard, not only refused life insurance, but as in the case the other day of a boy in one of the big schools, a big, husky boy, who had never been sick in his life, and who came up for the regular physical examination in the school. His whole family was thrown into terror because rales were heard at the apex, and the doctor who heard them said—"If that were my boy, he would not only not be allowed to go into athletics, but he would be taken out of school and treated as actively tuberculous for a year." Now that boy had nothing excepting rales at the apex, and the question is, what does that mean? In my experience more and more of such cases are coming along, and we are carrying in our tuberculosis clinics today hundreds, sometimes perhaps even thousands of cases, erroneously called cases of active tuberculosis, who are put there because of the fact that someone, perhaps keener than the average, heard something in that chest which was abnormal. Now, it is, of course, our job to hear those things, but it is also our job to interpret them, and you would be surprised to see how many people have such rales, which mean nothing more (and in autopsies you will see the pathological explanation) than a little thickened apex, a little thickening of the pleura and the underlying lung, with a little calcareous tubercle at the bot-

tom. No more is it proper to treat such a case as tuberculosis than to say that a person who has a scar of an appendicitis operation needs the operation. It is a question of the significance of the signs, and when you get signs of that kind it is a question of constitutional reaction. Now that is extremely difficult for the life insurance examiner, because a person who comes to be examined for life insurance is very apt to cover up and dissemble symptoms. When he comes to us as private physicians, it is with the idea of presenting all of his symptoms to help us make a diagnosis, so that I appreciate that the interpretation of signs is more difficult to a group like yours, than to us. At the same time we must bear in mind that, urged on by this tremendous anti-tuberculosis crusade, in which we are all proud to have a share, that we doctors have been driven to a point where we say that hitherto we failed in making early diagnoses and that we must make them, and consequently a good many of us have come to the point where we are now afraid not to make them, when ten or fifteen years ago we were making the mistake in the opposite direction of letting early cases slip by. Certainly, in my own experience it is true that of the errors that come to my attention from other practitioners, that I am able to detect (I am not speaking of those that I make myself), almost as many now are cases that are sent to me falsely diagnosed tuberculosis, as there are cases of tuberculosis that have not been properly diagnosed. Now, of course, the error and injustice is not so great, but from the standpoint of the scientific practice of medicine, it is really unsatisfactory, and the injustice through this is still great indeed.

Nevertheless, of course, when one cannot be sure, one can always watch, and that, I think, is important. We are, in my opinion, very much too prone to reach important decisions on the evidence of one examination, and if ever there is a disease which needs to be watched it is pulmonary tuber-

132 Thirty-Fifth Annual Meeting

culosis. We need to watch what the signs are, what the person does. And I don't know anywhere that it is easier than in a life insurance company, to defer decision, and see what the thing may be, and it is there that you are able often, if you are wise and not too impatient, to save yourselves doing great injustice to individuals and perhaps save the companies in which you may be interested a good many cases of good business.

There is another thing that I think is important in the question of diagnosis, and that is that tuberculosis of the lungs has a very definite way of coming in certain spots much more often than anywhere else. The upper lobe is, of course, the place. Generally speaking, localized signs with constitutional symptoms or without in the upper lobe are suspicious of tuberculosis, when similar signs in the lower lobe are not. The acute infections, the respiratory infections, of which we are learning more and more year by year, are much more liable to be in the lower lobes, and they often are localized and in their nature exactly the same as the signs of tuberculosis. I find it very helpful to think of the localization of tuberculosis in certain areas—certain points in the apex. There are four of them in front, and there are three of them behind. The first is, of course, above the clavicle. Then very frequently the signs are not heard above the clavicle, and you go down and listen perhaps with the stethoscope, and you hear nothing, because they go down in the central portion of the intercostal space, but curiously enough in these early cases, the signs are more marked in the outer and the third. Then another area is just outside the nipple. Those four places are places where I always listen, and if you do not get them there and get them somewhere else, the chances are they are not really tuberculosis. In the back, we have three places, just below the apex behind, a place which the French call the "Zone d' alarme"—just below the apex and just above the level of the spine of the scapula in the interscapular

space. Perhaps that is more common than anywhere else. The second place is the apex of the lower lobe which posteriorly is in the interscapular space, just below the level of the spine of the scapula. Then there is a place—and I think this is most important—that you cannot always hear unless you rotate the scapula by pulling the arm away forward, and that is, just along the margin of the scapula, down to about two inches above the angle of the scapula, and those are the lesions which go in from the root, along the interlobar fissure.

Those places are the ones we ought to look at with the greatest care and precision.

Lawrason Brown of Saranac has simplified this question of diagnosis a great deal. I think he has made a very real contribution to a subject which has been in danger of being tremendously overloaded by a lot of detail. You look through the literature as to the question of early diagnosis, and you will find that almost everyone who has contributed to the subject has the same bad habit of in some way over refining the methods of physical diagnosis in the matter of tuberculosis, until the ordinary man thinks—"My goodness! If we have to do all this and train ourselves to this point, early diagnosis is almost beyond us." As a matter of fact, early diagnosis ought to be within the range of the majority of cases of any well-trained general practitioner. Lawrason Brown has realized this, and he has set up five very simple criteria of what he thinks are cardinal signs, and he says that unless one or two of these are present, he is not willing to make a positive diagnosis of pulmonary tuberculosis, and he makes the statement that in the cases which he has analyzed he has only been wrong in 5 %, which he considers a pretty low margin. What are these five criteria?

1st. Tuberclle Bacilli in the sputum.

2nd. Localized rales above the third rib in front and above the spinal scapula behind, brought out of course by

the method of expiratory cough which is the only way you can really bring out the early rales of tuberculosis.

- 3rd. Parenchymous X-ray densities above the 3rd rib. The definite fluffy area with which we are all familiar.
- 4th. A definite indisputable history of frank haemoptysis.
- 5th. A definite, indisputable history of pleurisy with effusion.

Now, you can give that out to your examiners, because, as a matter of fact, it will come out right in nine out of ten cases, and it has been a very great help to have a man of Dr. Brown's position make the thing as simple as that. It is not of course always so simple, but in a great majority of cases it is, and I think it is a really positive contribution to simplifying this subject.

Differential diagnosis—the second point I want to emphasize, is more complex today than it has ever been before, because of the fact that we have more of the subacute and chronic infections of the lungs simulating tuberculosis since the influenza epidemic and a greater prevalence of that sort of respiratory infection in these past eight or ten years than we ever had before. When we speak of pulmonary tuberculosis and a differential diagnosis, we think often in terms of such things as chronic cardiac disease, in which the diagnosis was made because he happened to spit blood, and there is not much excuse for that faulty diagnosis—and yet, in my hospital service 25% of the many injuries were cases of chronic valvular disease of a mitral type, sent in as tuberculosis because they spat blood. Then there are the chronic conditions like chronic bronchitis, chronic abscess, then conditions which are rare, comparatively speaking, like cancer and syphilis. When we come to the question of these subacute respiratory affections, or influenza, or streptococci

or pneumococci, we are getting into a group which is often extremely difficult. I have been very much interested in these past eight or ten years in cases—and we have had a lot of them—that start in with influenza, or with a cough and respiratory symptoms and signs, and will persist in physical signs for months and sometimes even for years, but without any constitutional symptoms whatever. They are localized lesions, with rales and dullness and diminished respiratory murmur, but without much if any symptoms at all after the first week of onset, with very slight cough or expectoration, but with persistent signs over perhaps months or years. The main points in differential diagnosis are that the signs are so extensive, that if it is tuberculosis that patient is seriously ill, but if the patient has no constitutional symptoms whatever, and the sputum is always negative, and the X-ray changes are absolutely different from tuberculosis—in fact, almost nil, the case is probably not tuberculosis.

It is also important to remember that those cases are frequently associated with chronic infections of the upper respiratory tract—particularly chronic infected tonsils, chronic sinus infections, and that the chest will not clear up until the upper air passages are in good condition. Consequently, when you get cases of that kind with definite signs in the chest, chronic cough, no constitutional reaction, an X-ray and examination of the upper respiratory tract are essential from the standpoint of differential diagnosis.

I have been asked to speak regarding the X-ray. I am glad to do so, although it is not easy to discuss the X-ray with an audience without seeing the cases and the plates and going over them together. I would like to unburden myself, however, of a few ideas I have on this subject of the X-ray of the chest.

It seems to me in the first place that we are finally coming to a point after a number of years where we are considering the X-ray as part of the clinical evidence, that the

doctor who is to make a diagnosis in the case has got to take the X-ray into consideration just as he does auscultation. In other words, we are getting to the point where all of us must learn to interpret and read our X-ray reports in terms of the clinical findings, and not in terms of what somebody may think of that patient who has never seen him. We must learn to interpret X-ray plates of the chest, and the sooner we do that, the more accurately scientific we will be in our point of view.

The second point is that a great deal of evidence of value can be obtained with the fluoroscope. Many cases which have lesions that are important to differentiate can be differentiated with the fluoroscope. It is not an expensive method of examination. Where it is used a great deal it can be developed to a high degree of efficiency. I learned that in France during the war. Those French physicians were very clever in reading fluoroscopic screen shadows, and I learned a great deal from them. It is absolutely impossible to get much of value from a fluoroscopic picture in an early case of tuberculosis—you must have a plate. And although there is no doubt you can get a great deal of evidence from a flat plate, for final evidence in many cases you must have a stereoscope. This brings it all down to good technique, the question of opportunity for making these examinations, and then the question of interpretation, all of which does complicate the situation.

To summarize, I think we can use the fluoroscope more than we do, and where we cannot get the stereoscope we can get the plain plate, but where we can get the best, we ought to have the stereoscope.

There are a great many pitfalls in the question of the interpretation of the X-ray. I think it is here just as in the examination of the chest that we are often reading into the plate more than is really there. I know that all of us are getting more and more to believe that certain changes do not mean

tuberculosis, which we formerly thought did indicate it. The question of what is a normal X-ray plate is still a very much mooted question. The question of age and how much shadow you must allow as normal comes in. A perfectly definite typical case of pulmonary tuberculosis is something that can hardly be mistaken, although some cases of acute infection, like some of the broncho-pneumonias look very much like some forms of tuberculosis, but the ordinary tuberculosis at the apex, with its mottled fluffy appearance can hardly be mistaken for anything else, and consequently I think mistakes are not so often made in calling that kind of thing tuberculosis as in calling the little changes in tissues around the bronchi tuberculosis. We speak in general in the X-ray of three types of densities—

1. Hylus tuberculosis,
2. Peribronchial tuberculosis,
3. Parenchymatous tuberculosis.

Those are terms that more or less describe themselves, and it is the first two that are very much more difficult to diagnose. It is, in fact, doubtful whether one can make an X-ray diagnosis in hylus or peribronchial types, and I recall to your mind that Dr. Brown leaves out of his five points of diagnosis these first two. The parenchymatous densities are the typical ones.

Now, what does the X-ray shadow mean? When the X-ray man says that the laboratory reports what is apparently a healed lesion? Is it healed in a sense that it will not relapse? Is it healed in a sense that it is really not going to be a menace to that patient? I think the interpretation of such shadows is extremely important. In general, we believe that we can tell the main difference between a healed lesion and an active lesion, but there again, like in the physical signs—the X-ray is a help, and the physical signs

are a help but whether or not the disease is active at the time can only be told by the constitutional reaction of the patient, and that is what I want to urge more and more, that we have got to think of the question of the reaction of the patient to that particular disease, rather than the slight deviation from the normal in physical or X-ray examination. It is extremely important in X-rays to take your time, if you are in doubt. I do not know of anything more valuable than serial plates of a chest which you may suspect, or on which you are watching the progress of disease. If you have a case which you are not sure of, what is the harm in deferring it and not giving the patient insurance at once? If you can have an X-ray today and another next year to go with your physical examination, your evidence based on what has happened in the meantime is more than twice as valuable as what you get the first time. Consequently the X-ray is a thing which has its real place. I do not know of anything that has contributed more to the early diagnosis of tuberculosis in the last fifteen years than the general use of the X-ray. To me it is indispensable. I would no more think of allowing a patient to go out of my office without being X-rayed when there was even a suspicion of anything in the chest than I would think of allowing him to go out without examining his chest. Why? Because I have time and time and time again gone over a chest in which the symptoms may be suspicious, and heard nothing, and the X-ray showed definite evidence, and you put the X-ray and the symptomatic evidence together, and you have your diagnosis, and you have left the physical examination out of the picture altogether. On the other hand, there are cases in which the physical signs give positive evidence, and the constitutional reaction gives suspicious evidence, and they may have a positive sputum, and yet the X-ray is negative. These cases are rare.

If you take a hundred cases and have them X-rayed, then take the same hundred cases and have them simply examined,

there would be in my opinion fewer errors on the X-ray alone than on the physical examination alone.

Moreover, X-ray evidence if it is on a film or plate, is permanent evidence. It is there. You can pass it around and get somebody else's opinion about it. It is there as a record to compare with next year or the year after, which is of great value. Therefore, I should say that you cannot afford in life insurance work you cannot afford to allow the X-ray to get into the background. All cases which are suspicious in the first place, because of constitutional reaction, chest abnormal, weight below normal, or any of the suspicious signs which we are apt to have in our minds as possible symptoms of tuberculosis, if picked up by an X-ray will often help where the physical signs alone may be even negative, or at the best somewhat suspicious. So that I would like to emphasize my view that in life insurance work you are going to use more and more the X-ray rather than less. The question of interpretation of the healed lesion, and the question of what a certain shadow means in terms of duration of life, and consequently whether it is a good insurance risk is, to my mind, one of the most interesting and fascinating problems.

INSURANCE FOR ARRESTED CASES

I want to show you some of these slides now that will help to exemplify some of the things I have talked about. And then I am going to ask your indulgence in putting up to you a phase of the subject in which I am particularly interested, which I have not been asked to talk about at all, and that is the question of what you are going to do as medical men representing life insurance companies with the growing mass of people who have had tuberculosis and are now back at work, and who want life insurance. I know that there is a growing tendency among insurance companies to become more liberal, but there is still an attitude, I think, on the part of a good many companies something like this: These

cases are risks that we do not know much about, a patient once has had tuberculosis, the chances are that he is going to die of it sooner or later—we have enough good business, consequently we will turn his case down.

I see it from the standpoint of the patient whom I have watched for years, now perfectly well, who has his responsibilities, who wants to protect his family, and who cannot get life insurance, and the more I see it the more I believe that here is a great responsibility for you men. I can see it possible, if we knew enough, to give every apparently arrested case of pulmonary tuberculosis some form of life insurance, and I think we should work towards that end, and I believe it would be good business for the companies. Of course the trouble is, we don't know enough, and that is where the statistical studies have got to come in to help us. Certain it is that after I have seen a case live through every possible vicissitude of life, and every strain of life, and get well, over a period of three, four or five years, I would rather take that case, as far as the few signs in his chest are concerned, as a risk, than a good many others in which there were no physical signs at all, because that patient is leading a more or less sheltered life, he has learned the game of how to take care of himself, he is extremely careful in little things, and therefore as a rule he is less apt to have the other things that one may die of, than a person who has not had tuberculosis.

I would like to emphasize the question of time and of comparison. Would it not be possible to insure a case, for example, when apparently arrested, when he has signs of a healed lesion, has had no constitutional symptoms, is leading a normal life, after two years? Would it not be possible to make a special study of that class of cases and to say, we will consider such cases, of course at an increased rate? And would it not be possible each year to have them re-examined with the data right before us, with the X-ray, perhaps co-

operating with the medical man who has been following the case, and who perhaps could help the life insurance examiner come to a proper conclusion? Then each year as he kept well would it not be possible to diminish the extra rate and thus would it not be possible to do a great deal of good to a great many people about whom you do not see, as I do, the hardships that they have to go through, simply because they have the stigma of once having had tuberculosis? Is it not possible for us to contribute something to the welfare of those people by helping them to become self-supporting, to save money, and to protect their families from the various vicissitudes and accidents of life? Is it not possible that you gentlemen, representing insurance companies, could add a lot of really good business to your companies? I think you could, and I would like to suggest to you the possibility of making a more thorough survey than has yet been done of what happens to such cases very carefully selected, taking on trial a certain group, examining them carefully each year, and following them up on some such rated basis as I have suggested, and thus ascertain whether you could not accomplish a twofold good—to the patient and to the company?

Another point in that connection is, that we are coming to a new idea regarding the process of the healing of tuberculosis. We always thought that tuberculosis healed by fibrosis or by calcification. We know now that it often heals by resolution, that is the lesions clear up. Now if a case has resolved, as pneumonia would resolve, why should that case because he once had sputum and positive symptoms, be non-insurable, any more than a case of pneumonia? I am going to give you indisputable evidence that a certain percentage of cases of tuberculosis heal by resolution. Some heal partly by resolution and partly by fibrosis or calcification, but probably from 10 to 15% heal by resolution. That is a new idea—new to me a few years ago—and I have been watching it with a great deal of interest, and I think it gives a different

142 Thirty-Fifth Annual Meeting

slant to the question of relapse, and consequently of possible insurance risk.

I wish simply in closing to express my great appreciation of the opportunity of putting some of these things before you. I have tried to think out loud some on the things that puzzle me, and I wanted to try to stimulate your interest particularly because of the fact that I feel that your opportunities are very great and your responsibilities are correspondingly great.

Dr. Ward—Dr. Miller, we extend to you our hearty thanks for this splendid hour that we have enjoyed.

SECOND DAY

President Ward in the chair. The meeting was called to order at 9:30 o'clock.

The Secretary announced that he had cast a ballot as instructed for the election of the officers and members of the Executive Council placed in nomination on the preceding day, as follows:

| | |
|----------------------------|-----------------------------|
| President— | Dr. Chester F. S. Whitney |
| 1st Vice President— | Dr. Angier B. Hobbs |
| 2nd Vice President— | Dr. Wesley W. Beckett |
| Secretary— | Dr. Chester T. Brown |
| Treasurer— | Dr. Charles L. Christiernin |
| Editor of the Proceedings— | Dr. Eugene F. Russell |

Members of the Executive Council:

| |
|--------------------------|
| Dr. George A. VanWagenen |
| Dr. Edwin W. Dwight |
| Dr. J. Allen Patton |
| Dr. William Muhlberg |
| Dr. Ross Huston. |

These officers and members of the Executive Council were declared duly elected.

Dr. Ward—Dr. Bailey, of the New England Mutual Life Insurance Company will present to us a report on some work done by Dr. H. H. Amiral of that company on the normal diastolic pulse.

Dr. Bailey—Mr. President and Members of the Association—The chart which has been distributed was a result of some work done by Dr. H. H. Amiral, one of our Home Office Examiners, on the normal diastolic pulse. That is, a composite picture of the diastolic pressure taken under respiratory strain in forty-two young men. Many of you are familiar with Dr. Frost's work on the systolic pressure under respiratory strain, and this is a corollary of that work.

These forty-two young men were put through this test, Dr. Amiral taking the diastolic pressure on one arm and Dr. Frost on the other. Of course they were, so far as possible, simultaneous—that is, the minute that Dr. Frost got the highest point of the systolic, he released his armband, and Dr. Amiral then followed it down to the diastolic. There were presumably fifteen seconds between the two readings, but to all intents and purposes they were simultaneous.

They considered the diastolic pressure as being at the point where the systolic disappeared. There was no fourth phase. If you will look at the chart you will see that it is exactly the reverse method of the systolic pressure, the diastolic going up when the systolic comes down. Now, so far as we know, this is an absolutely unique observation. There is very little to be found in the literature on the diastolic pressure, and I think we all feel that it is important that we shall know what it means, and we do not know what this means. We have simply established this as a presumable norm. It is a composite picture of forty-two cases, and it is presumably normal.

We have come to believe that the systolic pressures give us the measure of the muscle of the heart, and we use the hypothesis that the diastolic may mean the condition of the

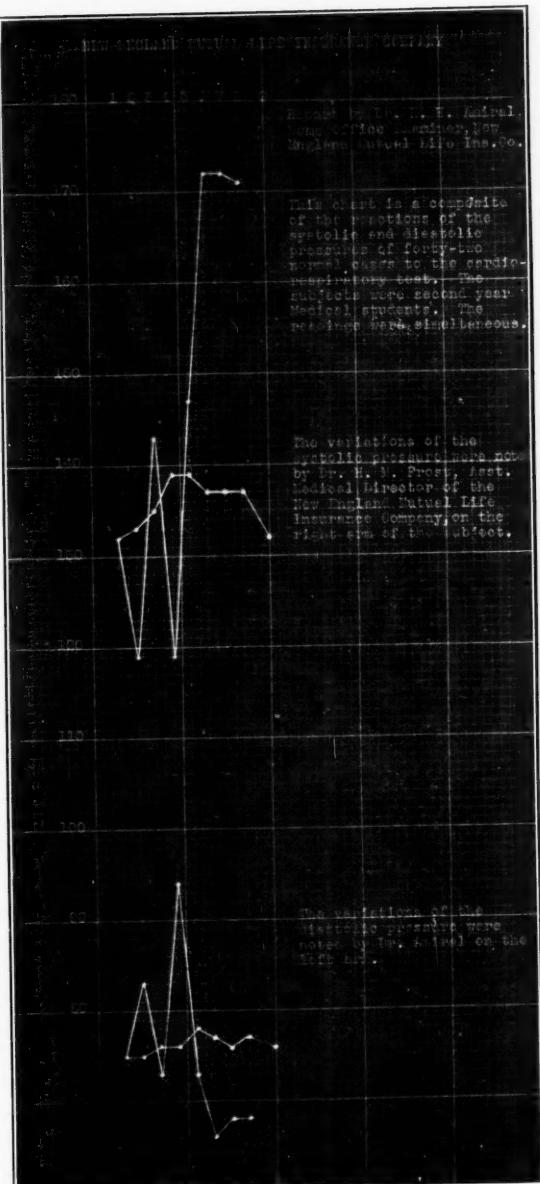
144 Thirty-Fifth Annual Meeting

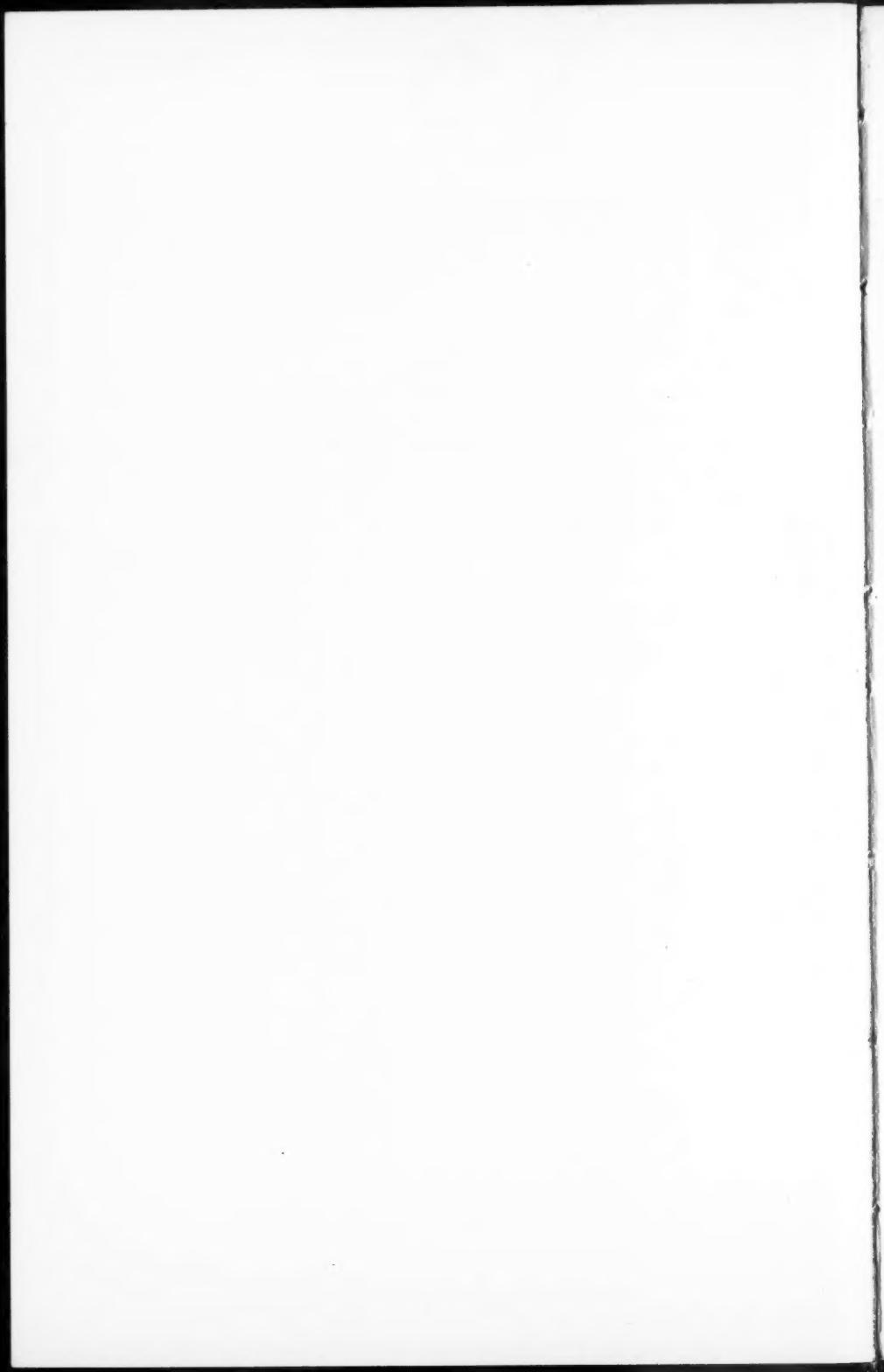
vessels, it may be a very sensitive measure of beginning arterial sclerosis. I have called that simply as a hypothesis, and we have only established this norm. We hope to begin on a series of abnormal cases and we hope to work out in the diastolic what Dr. Frost has worked out in the systolic, in different types of cardiac impairments. We report this simply as a foundation, and as something that we know at this time is absolutely new, and we hope some time later to make a further report on this diastolic pressure.

Dr. Ward—We will now take up the subject of the consideration of applications by lay approvers. I want to say that I feel a great sense of obligation to Dr. Huston for consenting to speak upon this subject. It was only a short time ago that I asked him to do so. It occurred to me that many companies have not organized a branch of this kind, and I know he will give us some very practical and sensible ideas. Following Dr. Huston, we will hear from Dr. Kanouse, of the Prudential.









Huston—Consideration of Applications 145

PRACTICAL METHODS FOR PROMOTING THE CONSIDERATION OF APPLICATIONS BY LAY APPROVERS

DR. ROSS HUSTON,

Medical Director Bankers Life Insurance Company.

For many years it has been the custom of certain large American insurance companies to handle some of their business received, through lay approvers and since a large percentage of the applications received by any company come well within so-called standard limits, few will doubt that it is a proper and practical way of handling the business. Experience has shown that it is safe because the mortality upon this business has been favorable. Having access to statistics, mortality tables and evidence of the past experience of other companies, the approving of this standard business, when properly safeguarded, has become mere clerical procedure.

In order that a successful department may be organized and maintained, it is necessary to choose as chief underwriter a man who has had considerable experience in the general laws and rules of insurance. He should be a man who has a sound educational foundation and a considerable knowledge of actuarial science. It is absolutely essential that he be a man of broad vision and willing to work in harmonious co-operation with the medical director of his company. He must be bold in backing up his own opinions and free to consult with the medical department in order to analyze the problems confronting him.

The choice of men for the positions of lay underwriters or approvers depends largely upon the class of business expected to be handled by them. Certainly the lay underwriter should be well acquainted with the office procedure and rules of his company. Those who have had the advantage of college

146 Thirty-Fifth Annual Meeting

work are preferable and they should have been in the employ of the company in the department of issue or in some department where they have learned the requirements of the company for standard insurance. It has been the practice in our own company to promote these men from our Policy Department or Department of Issue.

Proper training of these men is essential and placing them in a position to study their own job is necessary. By frequent conferences with the chief underwriter, medical directors, heads of departments and the other underwriters, the more experienced and qualified men soon become competent to handle certain business. It is usual to group them into junior and senior underwriters, according to the experience and efficiency of the men themselves. The so-called seniors are given authority to handle applications in amounts up to \$10,000, while the juniors are given authority to handle applications in amounts up to \$5,000. The work of the senior is checked by the junior as a part of the junior's training. These men approve the business according to established underwriting rules of the company. Proper limits are placed not only on amounts but their work is confined exclusively to cases coming clearly within standard limits, except those uncomplicated impairments of occupation, weight, moral and family history. They are given a chart showing the limit of the business to be handled by them and having had experience of one or more years in the Department of Issue, they know whether or not the case is one upon which they have authority to act.

We have found it practical to divide the territory in which we do business into five different districts. The division is so arranged that approximately an equal amount of business will be received from each district. We are training a lay underwriter for each district. By paying particular attention to his district he becomes acquainted with the agents writing the business, the character of the business which is presented,

Huston—Consideration of Applications 147

and familiarizes himself with the different doctors who are most active. These men are encouraged to prepare themselves particularly upon the hazards of certain occupations; for instance, the one who has charge of the territory in which the lumber and mining industries are most active may make a particular study of the occupations in this connection while another, in whose territory the manufacture of steel is the leading industry, may make a particular study of the hazards in this work.

In order to get first-hand information about occupational hazards in the different industries a personal investigation of many different plants has been made. Before we had a Lay Underwriters Department these investigations were made by the Medical Director, but since we have such a department this work is being done by the Lay Underwriters. The junior and senior men make such personal investigations as can be easily reached from the Home Office but it is the Chief Underwriter who makes trips into the territory to inspect the different plants. Some of the inspections which have been made of large industries are, the railroads, steel plants (including Bessemer and Plate Mills), rolling and wire mills, the glass industry, lumber and logging camps, some ore mines and smelters, knitting mills, telegraph and telephone occupations and the various branches of these different plants. The report of such an inspection is carefully studied by the underwriters and the medical department and comparisons made with previous reports and information which the company has. We have found the officers of these industrial institutions anxious that such investigations be made and they have given every help possible. We feel more competent to handle applications upon the lives of those in the industrial occupations after these personal investigations have been made. We realize that if we are to properly underwrite these risks we must keep in touch with the progress of the industrial trades. Safety engineers should be given credit for the fact that the

148 Thirty-Fifth Annual Meeting

hazards in accident and health of those employed by large industrial institutions are gradually but surely decreasing. An investigation of certain plants made five years ago is of little or no value today and we suspect that investigations of to-day will be of small value in a few years for the reason that different industries are constantly improving their working and living conditions. Every company writing substandard business or handling any considerable amount of occupational risks makes more or less of these personal investigations. It is expensive and after such investigation is made the results depend upon the experience of the inspector.

Why would it not be a practical thing to establish a Bureau of Industrial Investigation among the different companies for the purpose of establishing information at a *pro rata* cost? Each company could place their own interpretation upon the information received. I believe such a bureau would result in a more uniform rate on occupational risks.

Besides these personal investigations the lay approvers have access to a library containing modern books on occupational diseases and hazards, on industrial chemistry, the reports from the Department of Labor of both Government and State, and other industrial reports as well as such investigations as the company has been able to accumulate. They are encouraged to read scientific magazines and to learn as much as possible about the wage and living conditions of those in the industries.

Rules and regulations of underwriting cover in such a way the handling of those applications where the double indemnity is requested that this business is carried out as a matter of office detail.

We consider the underwriting of applications requesting disability benefit of much importance. Our own experience makes it necessary that we take great care in adding it to policies. Office rules cover the handling of these cases as to amount, height and weight, family history and occupation.

Huston—Consideration of Applications 149

About a year ago we added a question to our application asking for the income of the applicant from his principal occupation exclusive of his income from investments. We have a definite rule that we cannot grant disability to an applicant for any amount greater than two-thirds of his income. Of course the amount of disability carried with other companies is taken into consideration in arriving at the amount to which he is entitled. We do not grant the disability to young light-weights or to those with a tubercular history and since a large majority of the doubtful cases fall within these three impairments, the underwriters have no difficulty in handling most of them. Fully eighty-five per cent of the applications that we receive request one or both of the above features.

Moral and habit impairments require more conference between the underwriters and medical directors than any other. They usually go together and we have little data upon which to base an opinion. It is a matter of individual judgment. Little is known of the effects of modern prohibition and we cannot compare the class affected by it with those selected previous to the passing of the Eighteenth Amendment. More attention is paid to morals and finances in larger cases and since the amount handled by the lay approver is limited he is able to take care of a large percentage of the business. A case should not be rejected on a one man report and the period the reporter has known the applicant should cover more than one year. Unless the report covers more than one year and is from more than one informant, an alternate report should be ordered. All of this work is taken care of by the lay approvers. Doubtful risks should be sent to the Medical Directors or Risk Committee for action and such cases should be discussed with the lay approvers in order to give them the advantage of such decisions.

The impairment of family history is practically covered in home office instructions and the lay approver has no difficulty in handling this impairment.

150 Thirty-Fifth Annual Meeting

In the matter of weight, maximum and minimum limits are worked out and these cases are handled as a matter of clerical procedure so long as they come within these limits, otherwise they are referred to the Chief Underwriter who figures the proper rate.

Much of the correspondence with examiners, such as requests for additional information, is taken care of by the lay approvers. They also keep the agents advised as to the status of the case. This brings them in close touch with the field work which makes them more familiar with the problems of the different districts.

A definite period should be set aside two or three times a week for general review and conference where these men can ask questions about doubtful cases and where explanation may be given to them as to why approval or rejection is made. This close contact brings additional interest in their work. In these conferences there are three things which should be uppermost in the training:

1. That it is their duty to do everything they can to prove a case insurable before rejecting it.
2. To always leave the impression in every letter and conversation with fieldmen and officers of the company that their work is one of co-operation.
3. That the success of any business depends on the fairness of the decisions of those who are responsible for its growth.

Dr. Ward—Dr. Kanouse, of the Prudential, will open the discussion of this paper.

Dr. Kanouse—Mr. President and Members of the Association—Dr. Huston has covered the subject so thoroughly, there is not very much more to be said. Our plan differs somewhat from that which he has, inasmuch as the selection of the business side of the risk is left to the Issues Department almost entirely. We have striven to develop the men from the medical standpoint, and our plan is about the same as

Discussion—Consideration of Applications 151

theirs, so far as the grading of the men is concerned into senior and junior approvers, with one man in charge who is the referee. The others are graded as to their adaptability to the work, and the senior approvers act more or less in consultant capacities. They also train the new men whom it becomes necessary to add from time to time, as the responsibilities of the men are increased, and in the development of these men I think it is very important that you do increase their responsibilities. The selection of risks that are entirely normal on paper, with no deviation in the negative and affirmative answers, becomes a tedious one, and it adds to the interest of the workers to increase their responsibilities. When we started, we selected men from the Issues Department, feeling that they were more familiar with the work generally, and that it would be easier to train them than to select them from some other department.

They were first limited in the amount of insurance that they could act on and were permitted to approve only those cases that were uncomplicated by personal histories or the examiner's findings. It was soon recognized that there were greater possibilities in the use of laymen as approvers, and by placing them under the direct supervision of a medical man who gave them individual instructions and held conferences with them in which various answers to the questions appearing in the medical blank were discussed. A list of the commoner symptoms and diseases mentioned was made and instructions covering the action in each instance formulated. Soon these men became so proficient in this work that their responsibilities were increased until now they are acting on applications up to and including \$20,000 and are entrusted with certain classes of the substandard business. They are also permitted to reject applications. The codes, however, are applied by a second approver and the papers are finally reviewed by a medical man, who indicates his agreement with the action taken by affixing his initial.

152 Thirty-Fifth Annual Meeting

We conservatively estimate that the Lay Approvers of our Company pass on 85% of the business received.

Dr. Ward—I presume you have some questions to ask. The statement that the lay approvers of the Prudential are acting upon 85% of the cases is very interesting. Has any one any questions or any suggestions as to other methods to be used?

Dr. Ward—I would like to ask Dr. Huston one question. I gather that these lay approvers reject cases as well as approve them. When additional information or data is required, do the lay readers or their chief underwriter correspond with the examiners, or is that turned over to the Medical Board?

Dr. Huston—In doubtful cases, these approvers place their recommendation on the case, and it is sent to the chief underwriter. If it is complicated it comes to the Medical Department. If it is a purely uncomplicated occupation or weight impairment, they have the privilege of handling the case, otherwise it goes to the desk of the Medical Director. In fact, the rejections are double-checked before allowing them to become rejections, regardless of who makes the recommendation. These boys do carry on a large per cent of the regular inquiries to doctors, requests for additional examination, etc. In fact one of them who is classed as an approver, although he does very little approving, handles all or most of this part of the work, but in relation to the other men, they do handle a good deal of the correspondence themselves, not only with the doctors, but with the agents. The reason for this is that we want them to become familiar with the doctors in their particular territory, and with the agents in their territory, and also with the character of the business that comes from that territory.

Dr. Ward—I take pleasure in introducing Mr. Everett who, I understand, has charge of the lay approvers of the Prudential.

Mr. Everett—Gentlemen—We start from the point that the layman can perform quite a few functions in the selection

Discussion—Consideration of Applications 153

of risks and relieve the medical man, so that he can give his time to the specially complicated matters. The introduction of accidental death benefits and disability insurance has complicated the work a good deal, but in that field, too, we have found that the layman can serve. Whether the numerical system is used, or the selection is empirical, we think makes no difference in the function of the layman. Of course there is a distinct line, beyond which the layman cannot go, and that we think must be first impressed upon the laymen in their training. The layman is not a doctor. He can, however, review applications, and take final authority, and I believe unchecked authority, within reasonable limits, if he is properly trained and grounded. In our own company the history has been one of gradual and conservative growth. A few approvers were selected a number of years ago to act on small amounts, as Dr. Kanouse has said. Their work was limited entirely to so-called clean cases. Of course they covered the business side to start with, and a few impairments, build, and so on. That work was checked and for a period reviewed by medical men. They found it reasonably satisfactory, and gave the laymen further opportunities. The amount was increased and the impairments were increased. In the course of time we had the privilege of having an Assistant Medical Director associated with us for supervision purposes, and he saw the need for concentrating what we had done and giving us very definite instructions as a guide. Those instructions were used as the basis for increasing the number of approvers as we call them, and so the number of laymen engaged in the work and the quality of the work increased together. Under the supervision of this Assistant Medical Director, we had periodical meetings, sometimes, as I recall it, as often as once a week, and the medical officers gave us the benefit of their time, taking up the general subjects of various impairments, using our instruction book as a text, throwing the meeting open to questions, and getting some very sensible and wise

154 Thirty-Fifth Annual Meeting

questions, I believe. During the week, other questions would arise from special cases handled, and those we would save for the next meeting and discuss them there.

Following that, the growth of the business made it necessary for us to take on quite a lot of help in the lay end of the business. The problem was new men. We had not enough material in the ordinary new business department to select from. To some extent we had to go outside, but we have found laymen well grounded in the business adapted to this work, of a calibre higher than the average clerical standard, could be trained by intensive training, now done to a large extent by the laymen themselves, but of course with the constant medical contact. The location of some of our medical men on the same floor and practically in adjoining offices makes reasonable contact and co-operation very successful. They have been patient with us. We have used medical dictionaries and encyclopedias, and aids of that kind. I find that many of the laymen take so much interest in the work, particularly during their early training period, that they work nights, and study just as they would with a college or correspondence course. There comes the inspiration. It is an inspiration to be associated with medical men, and to see the direction in which selection is going, and their minds open to it. We depend upon them for a knowledge of the company's forms and plans, for bringing to the work a trained mind that will make consistent rulings, for accuracy, for care, within the scope of the layman, and we now have a staff of suitable size, with a supervising approver in charge. We aid in handling insurance inquiries also, and of course, both in the selection of new business *per se*, in rating for impairments, disability business, &c., in the final analysis, we realize that the complicated case or the case of an amount beyond our scope, is finally a medical question. We particularly watch for the cases where there are two or more related or non-related factors which finally come back to the question

Discussion—Consideration of Applications 155

of judgment anyway, and that judgment we realize must be medical.

In the rejection of cases because of moral hazard, the layman acts upon them, with the final O K of the medical men, but we find that lay approvers do good work there, and their recommendations are quite generally approved.

Dr. Cook—May I ask whether the correspondence in the Prudential with agents is also done by the lay underwriters, and if so, have they any experience that the agent is apt to feel that he should have the final authority from the medical man?

Mr. Everett—The lay approvers do write to the agents, but they write over the signature of the manager of the department of which we are a branch, in all matters other than medical matters.

Dr. Cook—On medical matters, a doctor signs the letters?

Mr. Everett—Yes.

Dr. Ward—Dr. McCrudden will now present his paper on the very interesting subject of Glycosuria.

156 Thirty-Fifth Annual Meeting

A PROPOSED METHOD OF SELECTING RISKS
AMONG INDIVIDUALS WITH OCCASIONAL
SLIGHT GLYCOSURIA.

By DR. FRANCIS H. McCRUDDEN,

Assistant Medical Director, New England Mutual Life Insurance Company.

I. INTRODUCTION

1. *Frequency. Seriousness.* In the experience of the New York Life Insurance Company the rate of actual to expected deaths among individuals with glycosuria was found to be 210 per cent of the medico-actuarial standard (373);* in the experience of the Metropolitan, 111 per cent of the American Experience standard (230). Among individuals with slight glycosuria on but one occasion the medico-actuarial investigation showed a mortality of 103 per cent of the expected (289). Barringer (25) found this impairment in 2,043 among 71,729 applicants, an incidence of three per cent. These examples illustrate what has been widely observed, that occasional slight glycosuria is a common impairment and that the group of individuals with this impairment is, as a whole, uninsurable at standard rates. The question arises: How shall these individuals be dealt with by the insurance companies?

2. *Characteristics of this group.* This group has two striking characteristics:

(1) Its mortality is greater than the standard;

(2) The group is heterogeneous with respect to attributes which influence mortality.

Either of these characteristics may be made the basis of a system for dealing with these individuals.

*These bracketed numbers refer to the references of corresponding numbers at the end of the paper.

3. *Sub-standard insurance.* A higher mortality than the standard need offer no bar to insurance. If we know the mortality of a group we can insure its members by charging such premium rates as the higher mortality calls for. This system of dealing with groups of individuals having impairments may be called *sub-standard insurance*.

4. *Homogeneity of groups.* The basic principle of another system for dealing with this problem can be traced to the astronomer Halley. In 1690 the British government attempted to raise a loan of one million pounds by issuing annuities, the annuities being made payable at the rate of fourteen per cent per year on the assumption that the average expectancy of life for the population as a whole, irrespective of age, was seven years. In 1693 Halley pointed out the practical error of assuming a rate for a group so heterogeneous with respect to age, an attribute which so profoundly influences mortality; and from the bills of mortality of the city of Breslau he thereupon constructed a mortality table and formulated a method of calculating insurance rates for different age groups (485). Halley's table and formula form the basis of modern life insurance rates.*

5. *Physiological and pathological homogeneity.* But although the fundamental idea of dividing people for insurance purposes into groups homogeneous with respect to attributes potent in influencing mortality and then actually determining the group mortality is implied in Halley's mortality table over two centuries ago it seems to have been applied only to physiological attributes, age and sex (486, 487) for example. Up to 1905 there is no record in the literature of an application of this principle to the determination of the influence on mortality of a pathological attribute. On page 446 of the Transactions of the 16th annual meeting of the Association of

*Footnote. The first mortality table, published by Graunt in 1662 (484) was too crude to be of any practical use, and was not constructed with any reference to life insurance.

158 Thirty-Fifth Annual Meeting

Life Insurance Medical Directors in 1905 Dwight, Medical Director of the New England Mutual Life Insurance Company, after defining a certain class of risks (the group with a history of some renal impairment who are able at some time to pass a normal specimen of urine) states, "During this time" (2 years) "we have accepted 785 risks of this class. According to the American Experience Table we should have expected a mortality of 6.8%. One of these risks has died up to this time * * *" (96). This statement is the *Anlage* of the whole modern science of medical selection.

6. *Medical selection.* The principles of this system may be summarized as follows. The mortality of any group of individuals depends on certain physiological, social and pathological attributes, such as age, occupation and state of health, of the members of the group. Any heterogeneous group—and every group of more than one member is heterogeneous in some respect—may be divided into sub-groups each relatively homogeneous with respect to one of these attributes; and each such sub-group will have its own mortality. A heterogeneous group uninsurable at standard rates may be divided into sub-groups in this way until we find a sub-group with a mortality less than the standard. This system of dealing with individuals having impairments is the system made use of in this paper and may be called *medical selection*.

7. *Methods of selection.* There are three general methods of picking insurable subgroups of individuals from uninsurable groups; the inductive method, the deductive method, and the method of over-selection.

8. *Inductive method.* The group may be subdivided on the basis of age, height, weight, occupation, and other characteristics of which we have a record and the extent to which the characteristic influences mortality may be determined by computing the mortality of the subgroups. For example, the mortality of the group subdivided according to age, as

reported by Knight (230) is shown in table 1 and as reported by the medico-actuarial investigation (289) in table 2. From the figures it is evident that the subgroup between the ages of about 30 and 40 is, possibly, insurable at standard rates. This may be called the inductive method.

Table 1

The Mortality of Individuals With Glycosuria on One Occasion Subdivided According to Age (230)

| Age Group | Ratio of Actual to Expected Deaths |
|-----------|------------------------------------|
| 15-24 | 120 |
| 25-34 | 88 |
| 35-44 | 99 |
| 45-54 | 124 |
| 55-64 | 118 |
| Over 65 | 222 |

Table 2

The Mortality of Individuals With Glycosuria on One Occasion (289)

| Age Group | Ratio of Actual to Expected Deaths |
|-------------|------------------------------------|
| 20-24 | 100 |
| 25-29 | 134 |
| 30-34 | 63 |
| 35-39 | 87 |
| 40-44 | 116 |
| 45-49 | 103 |
| 50-53 | 110 |
| 54-56 | 175 |
| 57-59 | 107 |
| 60 and over | 67 |

9 *Deductive method.* We may use as the basis of subdivision some characteristic of which we have no record in our old examinations but which our medical knowledge leads us to suspect may profoundly influence the mortality; we may then insure the subgroup selected on this basis and subsequently determine the mortality. This task may be divided into three parts:

- (a) the division of this group of individuals into two smaller subgroups more homogeneous with respect to attributes fundamental enough to warrant the assumption that they probably influence mortality,

160 **Thirty-Fifth Annual Meeting**

(b) the identification of members of these subgroups,

(c) the determination of the mortality of the subgroups.

Scrutiny of the causes of death in risky groups may give a lead as to attributes which are particularly significant in such groups; on account of the increased mortality from circulatory disease among overweights, for example, we pay particular attention to the condition of the kidneys and heart in such cases. This may be called the deductive method.

10. *Over-selection.* Finally there is a method of selection based on the fact that the mortality of individuals above the average in all characteristics which influence mortality—the "excellent" group—is less than that of our risks as a whole—the "standard" group. Taking advantage of this fact we may assume that the lowering of mortality due to "excellence" will offset the increase in mortality due to one minor impairment and on this basis accept individuals with one minor impairment who are above the average in all other respects. This may be called the method of over-selection.

11. *Comparison of methods.* The inductive method gives results which can be predicted but it is limited in application because some characteristic not recorded in our old policies may so profoundly influence the mortality of a group that in comparison with this unknown characteristic those which we do have on record are practically negligible. The results of the deductive method are uncertain but its possibilities are unlimited. In practice we try the inductive method first; when we have exhausted the possibilities of subdivision according to attributes which we have on record we may then try the deductive method. The method of over-selection is a last resort; it is not specific enough; it leads away from scientific group selection and tends toward the old clinical method of individual selection.

12. *A Proposed method.* Studies of the mortality records of the group of individuals with occasional slight glycosuria show no characteristics on record in our old policies which influence mortality enough to make them the basis of a satisfactory subdivision of the group. In practice the method of over-selection has been considerably used (118, 341, 342, 44). But our knowledge of carbohydrate metabolism has increased rapidly in recent years and there is reason to believe that we can offer a promising provisional method of selection based on deductive reasoning. The facts on which we have based such a method follow.

II. DIABETIC AND NON-DIABETIC GLYCOSURIA

(a) GLYCOSURIA IN DIABETES

1. *Willis.* The first contribution to the literature of glycosuria, the observation that enabled us to distinguish diabetes from other forms of polyuria, was made by Willis (1674) who pointed out that the urine in this condition is sweet (455, 466):

"But as to what several authors say, that the drink is little or nothing changed, there is no truth in their assertion; because in all people that I have ever happened to know, and I believe it is so in all, their urine was very different not only from the drink that they took in but also from any other humours that are usually generated in our bodies, being exceedingly sweet, as if there had been sugar or honey in it."

2. *Dobson.* A century later Dobson (91) isolated from diabetic urine a sweet fermentable solid. He says:

"Two quarts of this urine was * * * evaporated * * * to dryness. * * * There remained after the evaporation a white cake which weighed IV drachms. This cake was granulated and broke easily between the fingers; it smelled sweet like brown sugar, neither could it by the taste be distinguished from sugar except that the sweetness left a sense of coolness on the palate." * * *

"On letting the diabetic urine stand, it fermented—on shaking the vessel a vinous smell was easily distinguished. Soon

162 Thirty-Fifth Annual Meeting

after this the fluid became sourish * * * the next change was to the keen smell of vinegar."

3. *Cawley.* In 1788 Cawley (73) says of a diabetic, "his urine was found to be sweet, fermentable with yeast, and two pounds, on evaporation, yielded about five or six ounces of sweet black extract exactly resembling that preparation of molasses made by confectioners for children, and vulgarly called coverlid." In carrying out an autopsy on this patient Cawley noted pathological changes in the pancreas whose significance, naturally, he did not appreciate. As this first reference to pancreatic disease in diabetes seems to have passed unnoticed, so far as I know, it seems worth recording:

"The pancreas was full of calculi which were firmly impacted in its substance. They were of various sizes, not exceeding that of a pea, white and made up of a number of lesser ones, which made their surface rough, like mulberry stones; and in all respects they appeared analogous to the calculi which we sometimes meet with in the salivary ducts. The right extremity of the pancreas was hard, and appeared to be scirrhouss."

The only reference to this autopsy that I have come across does not mention the pancreatic changes; Salomon (382) in a history of the early literature of diabetes refers to it as follows:

"Except for an unusual paleness and softness of the kidney, the autopsy showed nothing abnormal."

4. *Identification and determination.* In 1806 Dupuytren and Thenard (95) obtained alcohol and carbon dioxide by fermenting diabetic urine demonstrating, thereby, that the sweet substance in diabetic urine has the properties of real sugar. In 1815 Chevreul (75) isolated this substance in crystalline form and identified it as grape sugar. With the introduction of Trommer's qualitative test for glucose in 1841 (426) and Fehling's quantitative test in 1848 (110, 111) the examination of urine for sugar became a routine chemical procedure.

(b) DIABETIC TRANSIENT GLYCOSURIA

1. *Early work.* As long ago as 1884 Frerich (133) recognized that transient glycosuria may be the first sign of diabetes. In 1891 Kraus and Ludwig (234) emphasized the seriousness of occasional traces of sugar in the urine. In 1899 Raphael (364) reported a case of transient glycosuria which developed into real diabetes. In 1906 Naunyn (324) pointed out that in early diabetes sugar may appear only intermittently, after hard work or after the ingestion of certain foods.

2. *Von Noorden.* In the 1917 edition of his book Von Noorden (329) refers to nine patients, among them four physicians and two chemists who were in the habit of testing their urine at intervals long before the appearances of glycosuria and who could, therefore, tell him exactly when glycosuria first appeared. In not one of them were there any symptoms either at the time sugar first appeared or for months afterward. He refers to 24 diabetic patients who had occasional traces of sugar in the urine at times from five to fifteen years before the onset of any other symptoms suggesting diabetes.

3. *Joslin.* Joslin (218) mentions specific cases (p. 471):

"In many cases the disease breaks out temporarily before the glycosuria becomes permanent."

"Case #129 showed sugar in the urine in 1901 at the age of three years at a time when she appeared out of condition. Examining frequently after that I failed to find it and did not look for it again until February, 1905, when she appeared like a full fledged case of diabetes. Death came on in July, 1907."

"Case #235 showed 1.3 per cent sugar in the urine January 3, 1901, at the age of twenty-six years one month after an attack of severe catarrhal jaundice. The glycosuria disappeared at once on restriction of diet and did not return after resumption of a liberal diet containing sugar, December, 1904,

right pyelonephritis, urine sugar-free; January 3, 1905, sugar appeared with a moderate amount of acetone, but no diacetic acid, and the patient became sugar-free with a strict diet and, until July, 1906, was able to eat freely of toast, oatmeal, potato, rice and sugar without glycosuria. Death in coma, May 4, 1910."

"Case #1008 showed sugar in the urine on repeated occasions at the age of forty years, was carefully treated for sixty days and later no sugar was found, but it reappeared when the patient was fifty-one and he came under my observation three years later, with severe diabetes."

Joslin's conclusions express his general experience with these cases.

"An individual who at one time had been diabetic or even had a suspicion of glycosuria which has passed for years as cured and has even dropped out of memory, perhaps for decades, may again become diabetic under favoring influences."

"I am always very slow to tell a patient he does not have diabetes if any doctor has ever found sugar present in the urine or thought it present."

4. *Normal urine in severe diabetes.* Even in severe diabetes, when properly treated, the urine may be free from sugar for long periods. And even without treatment patients with severe diabetes and high blood sugar concentration may show little or no glycosuria. In case #VI (see table 4), a very severe case of diabetes, the urine showed no sugar. One hour after ingestion of 100 grams glucose the urine contained only 0.16 per cent sugar, an amount that would probably be missed with the usual tests for glycosuria.

(c) NON-DIABETIC TRANSIENT GLYCOSURIA

1. *In disease.* But transient glycosuria may occur in conditions other than diabetes. It has been reported as the result of many acute infectious diseases; cholera (63); diphtheria, scarlet fever, typhoid fever, measles, appendicitis, influenza, and other infectious diseases (65); malaria (72) (173);

mumps (239); anthrax (353); renal tuberculosis (402); and hyperthyroidism (407). Slight transient glycosuria may result from chronic disease; obesity (394), and hypertension (269). It has been reported as the result simply of old age (394).

2. *From drugs.* Drugs and other chemicals can cause glycosuria. Epinephrin (47, 48), phlorhizin (290), salt injections (50, 235), ether anesthesia (176), alcohol (64), morphin (252, 253), chloral (252, 253), carbon monoxide (175, 366), hydrocyanic acid (133), sulphuric acid (133), ortho-nitrophenylpropionic acid (192), caffeine (195), theobromin (195), diuretin (195), uranium (275, 276), cantharides (367, 368), zinc (381), corrosive sublimate (389) and mushroom poisoning (4) have all been known to cause glycosuria.*

3. *From cooling.* Glycosuria has been observed in dogs and rabbits (10) and in man (148) as the result of exposure to low temperature. Moderate cooling of the gastro-intestinal tract following opening of the peritoneal cavity causes glycosuria (23).

4. *Alimentary glycosuria.* There seems to be a general impression that ingestion of excessive amounts of sugar can cause glycosuria; but the subject has not been much studied. Von Noorden (329, footnote p. 331) refers to a sugar factory in which the workers are allowed to eat all the sugar they want. The new workers make full use of the privilege with the result that in the first two or three weeks sixty per cent of them show a very slight (one to two per cent) glycosuria; later the glycosuria disappears. Marsh (278) has described one case of alimentary glycosuria following average normal meals which on further study turned out not to be diabetes.

*FOOTNOTE—The literature on drug glycosuria is very extensive; I have referred here to only the first observers in each case.

166 Thirty-Fifth Annual Meeting

5. *Nervous glycosuria.* Glycosuria is a common sequel of injuries or other changes in the central nervous system. Bernard (39) observed it after section of the vagus and after puncture of the floor of the fourth ventricle. It has long been known as a sequel of apoplexy (250, 133, 163), Naunyn (324) refers to the occurrence of glycosuria as the result of brain tumor, and injuries and diseases of the sympathetic. Lépine (247) cites many cases of glycosuria following lesions of different parts of the nervous system. In general paresis Straus found glycosuria in 5 out of 57 cases (407); Bond found it in 3 out of 30 cases (54); Siegmund found it in 10 out of 79 cases on the first observation—thirteen per cent—in eight per cent more on the second observation, and in fifty-two per cent on repeated observations (403).

6. *In psychoses.* Glycosuria is a common accompaniment of psychoses. Laudenheimer (241) found it in 30 out of 1,250 insane patients (2.4 per cent). Among 315 insane individuals Siegmund (403) found it in more than eight per cent. Dawson (87) found glycosuria in seven cases of mental disease whose predominating characteristic was "mental discomfort." Folin (125) found glycosuria in 22 out of 192 insane patients, the great majority of whom suffered from depression, apprehension, or excitement. Mita (304) observed glycosuria in 8 out of 35 cases associated with depression. Schulze and Knaue (391) found sugar in 150 out of 2,500 specimens of urine from insane patients. Two-thirds of the specimens showing sugar came from patients in the melancholic stage of manic-depressive insanity; with a remission or a cure the sugar disappeared from the urine. This investigator rarely found sugar in the urine of sociable good-natured patients. Schulze (390) reports glycosuria as common in patients with depression, no matter what causes the depression; he found it even in individuals who were apparently normal aside from a feeling of depression, and

noted that with disappearance of the depression the glycosuria cleared up. He noted further that the greatest amount of sugar is excreted in the fear psychoses and that the amount of sugar in the urine varied with the severity of the condition.

7. *In mental depression.* Goodhart (151) especially has called attention to the association of glycosuria with attacks of nervous depression. A few of his observations are worth citing:

"He came to me in a deplorably wretched state of mind, trembling all over, and half sobbing, as he detailed his varied feelings * * * was always nervous; but that now he was especially knocked down by having lost his senior in a bank."

With recovery sugar finally disappeared.

"Another man * * * holding a leading and very anxious position * * * in addition to all this worry involved * * * had a lot of domestic anxiety * * * irregular appearance of sugar * * * none when I saw him last for several days."

"Another case of decidedly neurotic type * * * if anything happens to him either in business or pleasure his nerve tone collapses and he becomes cowed and depressed quite beyond recognition * * * I wish it were possible to convey to your mind's eye the picture of the two sides of this man, big, jovial, bouncing when he is well; slouching, drawling, despondent, limp in mind and body when he is glycosuric * * * had seen him never with glycosuria till one day he came to me saying he had been quite knocked over by worry in business and he quite looked it. If ever there was a dog with his tail between his legs it was he * * * by this means had knocked his nerves up completely * * * afraid of meeting anyone; a sudden noise would startle him, make him quake * * * urine contains sugar." * * *

Later on the urine became normal. Goodhard describes several other cases of what he calls "paroxysmal neurosis"—intermittent attacks of melancholia accompanied by glycosuria followed by healthy spells without any glycosuria.

168 Thirty-Fifth Annual Meeting

8. *From fear.* In healthy persons, also, and even in animals emotional stress leads to glycosuria. In 1877, Böhm and Hoffman (52) reported that cats showed glycosuria simply as the result of being tied down to a board; the discoverers called this "hobbling" glycosuria. By eliminating all pain in the process Cannon, Shohl, and Wright (69) demonstrated that hobbling glycosuria is due to fear and not to pain. The few cats that accepted this process calmly showed no glycosuria. By demonstrating sugar in the urine of cats which were badly frightened by being placed in cages near barking dogs these authors completed the proof that fear is the cause of the glycosuria. Even the cats that were not frightened and, therefore, showed no hobbling glycosuria showed fear glycosuria when frightened by the dogs. It has been demonstrated by Jacobson (196) that the hobbling glycosuria of rabbits is, likewise, due to fright.

9. *From apprehension.* Continuing his studies in man Cannon (68) observed glycosuria in 12 out of 25 members of the Harvard football squad immediately after the most exciting contest of the season. Five of the twelve were substitutes not called upon to play. The only specimen of urine from a spectator showed marked glycosuria. Hammett (169) found that 9 out of 12 football players (57 per cent) developed glycosuria as the result of participation in a decisive game. 6 out of 7 substitutes (85 per cent) developed glycosuria as the result simply of watching the game with the possibility of participation. 6 out of 13 mere spectators developed glycosuria. Cannon (68) observed glycosuria in 4 out of 79 students (all normally aglycosuric) as the result of a hard professional examination. Hammett (169) found sugar in the urine of 11 out of 27 students (43 per cent) immediately after a short but difficult written examination; none of them had shown glycosuria before the examination. The effect was transitory; only one of the eleven still showed glycosuria four hours later.

Folin (125) examined the urine of 34 second-year students before and after an examination; one showed glycosuria before and after the examination; of the remaining 33, 6 (18 per cent) showed glycosuria after the examination only. In another series, out of 36 students taking an examination, none of whom showed glycosuria the day before, 6 (17 per cent) showed glycosuria immediately after the examination. Worms (468) found sugar in the urine of 10 out of 100 persons engaged in sedentary occupations involving mental activity; but he did not find glycosuria in a single one of a series of 607 laborers. Joslin says (218):

"Case No. 511 shows sugar when hard at work in the city, but when quite as occupied with mental work in the country, but with more exercise and a similar diet, shows no sugar."

(d) SUGAR IN NORMAL URINE

1. *Early work.* Brücke (59, 60, 61, 62) using the reduction test and the fermentation test, was the first to allege the presence of traces of glucose in normal urine. Shortly afterward Tuchen (427), using the same tests and the polariscope as well, confirmed Brücke's findings. By using large quantities of urine Pavy (345) was able to get results by reduction and fermentation which seemed to confirm each other. Müller (315), Nylander (331), and Quinquaud (358) showed that a large proportion of the specimens of urine which show a slight reducing power before fermentation with yeast, do not reduce after fermentation; the inference being that it is glucose, later destroyed by fermentation, which causes the reduction. Wedenski (444) isolated glucose from normal urine in the form of its benzoyl ester and identified it by elementary analysis. Rosin (377) later repeated and confirmed the finding of Wedenski. Several years after Fischer had shown that sugars can be identified by their osazones (115), Moritz (309) prepared glucosazone from normal urine; this was repeated and confirmed by Baisch (16, 17).

2. *Quantitative determination.* Investigations of the quantity of sugar in normal urine are open to the criticism that there is no practicable quantitative test that is specific for glucose; other substances in the urine respond to the quantitative tests in use or interfere with their accuracy. The various tests with alkaline copper sulphate are merely tests for reducing substances. Lactose, maltose, levulose, pentoses, and homogenetic acid (occurring in cases of alkapttonuria) may be responsible for a positive reduction test. Drugs, especially the quinone derivative cathartics, may cause reduction. Too high figures may be due to kreatinin or uric acid (265). About half the reducing power of normal urine is due to kreatinin and uric acid (242). A considerable proportion of the reducing power of normal urine is due to urochrome (53, 21). Too low results may be due to the fact that kreatinin can hold some of the reduced copper oxide in solution (265). Ammonia produced from the urea on heating also helps hold the cuprous oxide in solution (36). When urine is boiled with alkaline copper solution, some of the copper is first reduced by the glucose and uric acid; if the amount of reduced copper is small, it is held in solution or suspension by kreatinin. On continuing the boiling, more of the copper is reduced by kreatinin which, as the result of this action, is itself destroyed so that, in time, there is not enough kreatinin left to hold the increased amount of cuprous oxide in suspension and it precipitates. The polaroscopic method, likewise, is open to criticism: glycuronic acid as well as glucose is dextrorotatory; and levulose, β -oxy butyric acid, and the compound glycuronates are laevogyatory. Levulose gives the same osazone as glucose; and maltose and isomaltose, which occur in normal urine (16, 17) (245, 265), give an osazone with a melting point which differs from that of glucosazone by only one degree. Furthermore, the glucose, giving a glucosazone, might be a secondary product formed from something else in

the process of preparing the glucosazone. As for the fermentation method, the difficulty of obtaining a yeast that gives a negative control throws doubt on conclusions based on that method (478).

3. *Recent work.* Various methods for removing the interfering substances from the urine have been devised; mercuric nitrate (388, 35), phosphotungstic acid and lead acetate (336, 335), picric acid (320, 321), blood charcoal and 25 per cent acetic acid (375), 15 per cent acetone (272). With the aid of such methods the whole question of glucose in normal urine has recently been reinvestigated by Benedict of Cornell and Folin of Harvard. According to Benedict (35, 36), after removal of interfering substances, normal urine shows a reduction (of picric to picramic acid) corresponding to 0.7 to 1.10 grams glucose a day. The amount is increased by ingestion of a diet rich in carbohydrates and may reach an upper limit of 1.6 grams a day. Benedict alleges that the difference between normal and diabetic urine is merely quantitative. After repeating and extending Benedict's investigations Folin and Berglund (124) admitted the presence of a reducing substance in normal urine which is slightly increased in quantity by the ingestion of large amounts of vegetable carbohydrate food; but they observed that it is not at all increased in quantity by the ingestion of pure glucose and concluded, therefore, that it is not glucose but some other reducing substance of a dextrin-like nature. It is evident that the question of the glucose content of normal urine has not yet been settled to the satisfaction of everyone. As the matter stands now, normal urine seems to contain small traces of reducing substances but little, if any, glucose; from which the conclusion might be drawn that a too highly sensitive test for reduction is less useful for practical purposes than the less sensitive Fehling's test.

III. THE CAUSES OF GLYCOSURIA

(a) BLOOD SUGAR

1. *Dobson.* As long ago as 1774, Dobson (91) asserted that the sugary substance eliminated in the urine in diabetes is not formed in the kidneys, but comes from the blood:

"Experiment II"

"Eight ounces of blood taken from the arm * * * was sweetish, but I thought not so sweet as the urine."

"From experiment II it appears that this saccharine matter" (in the urine) "was not formed in the secretory organ but previously existed in the serum of the blood."

He concluded that diabetes is a species of imperfect digestion, or assimilation, a disease of the system in general.

"This idea of the disease also will explain its emaciating effect; from so large a proportion of the alimentary matter being drawn off by the kidneys, before it is perfectly assimilated, and applied to the purposes of nutrition. The diabetes proves, in some cases, a very rapid consumption."

Cullen (82), later claimed credit for this hypothesis (*p. 91*):

"I think it probable that, in most cases, the proximate cause of this disease is some fault in the assimilation powers in converting alimentary matters into the proper animal fluids. This I formerly hinted to Dr. Dobson, and it has been prosecuted and published by him."

2. *Bernard.* During the first half of the nineteenth century a number of investigators searched for sugar in diabetic blood. The results of these investigations are of doubtful value and difficult to appraise because of the imperfect technique used. Normal blood was not studied because it was believed at this period that unless sugar is ingested normal blood does not contain any. Physiologists were still dominated by the old idea of Lavoisier that animals can only oxidize

organic compounds, that only plants can synthesize and that animals utilize the products synthesized by plants. The proceedings of the French Academy of Sciences in the 40's contain papers by such eminent chemists as Boussingault and Dumas contending that all the butter fat and lactose in cow's milk, for example, is preformed in the vegetable food. But Claude Bernard did not accept the prevailing opinion. He searched for and found glucose in the heart's blood of normal dogs and rabbits on various diets and even when they were starving (39). He was unable to find sugar in the portal vein, however, and came at first to the erroneous conclusion that the sugar is formed in the liver (40). Bernard's assertion that the blood contains sugar was contested for years. Ritter (371) insisted that the sugar found by Bernard was formed post-mortem in the liver and then diffused into the blood. It was not until about 1870 as a result of a series of investigations in several laboratories that Bernard's findings were generally accepted; and it took much longer to remove all doubt.

3. *Fiquier.* As early as 1855 Fiquier (113) went further than Bernard and asserted that he could demonstrate sugar in the blood of the general circulation. Bernard denied the correctness of Fiquier's assertion (41). The controversy provoked so much discussion that the French Academy appointed a committee to investigate and report. The committee reported the presence of a reducing substance in the blood but denied that it was sugar (*Comptes rendus* June 18, 1855). Fiquier (114) answered this objection by showing that he could ferment the sugar and produce alcohol.

4. *Specific identification.* In 1884 Seegen (393) showed that quantitative sugar determination by Fehling's method, by polarization, and by fermentation in blood from which all protein had been removed gave results in practical agreement on the assumption that the sugar is glucose. This was con-

174 Thirty-Fifth Annual Meeting

firmed by Pickhardt (354). In 1890 Moritz (309) prepared an osazone from blood which he identified by its melting point as glucosazone. The preparation of glucosazone from normal blood has since been repeated by Hedon (177), Hanriot (170) and Pickhardt (354).

(b) GLYCOSURIA WITH HYPERGLYCEMIA

1. *Methods.* It is only recently that reliable and clinically practicable methods of measuring the amount of glucose in the blood have been developed. In all methods now in use the reducing power of the deproteinized blood filtrate is used as a measure. Various precipitants have been used for removing the protein: sodium sulphate and dilute acetic acid (372), alcoholic zinc acetate (1), mercury chloride (386), phosphotungstic acid (443, 126), colloidal iron (374, 398, 267), dialysis against isotonic salt solution (98), alcohol followed by colloidal iron (106), iron alum (439), alcohol and animal charcoal (22), picric acid (256, 32, 33, 102, 350, 271, 320, 321, 83, 2, 452, 226). The glucose in the filtrate has been measured by reduction of the copper and determination of the excess of copper by titration with hydroxylamin (20, 220), or by determination of the reduced copper by weighing (443), by solution in ammonia (398), by titration with ferric sulphate and permanganate (483) by changing the copper to colorless cuprous sulphocyanate and determination of the excess of copper sulphate colorimetrically (13), titration of the cuprous oxide with iodine (22, 400, 266, 267), by redissolving the cuprous oxide in phosphomolybdic acid with production of a color which can be quantitatively measured (126, 127, 29, 307, 233). The glucose has been determined by colorimetric measurement of the amount of picric acid that will be reduced to picramic acid by a definite quantity of deproteinized blood filtrate in the presence of sodium bicarbonate (256, 32, 33, 102, 350, 271, 320, 321, 322, 83, 2, 452, 226). Wacker has made use of the color reaction between glucose and para-

phenylhydrazinsulphonic acid (439) and Reicher and Stern (365) have made a quantitative application of Molisch's reaction. Rona and Michaelis have determined blood sugar quantitatively with the polariscope (374). Methods in which picric acid is used as the oxidizing agent give results slightly higher than the other methods but otherwise different methods show comparatively close results (120, 90, 308, 79, 185, 81, 420, 413).

2. *In diabetes.* As long ago as 1878 Bernard (42) suggested that diabetic glycosuria is due to high blood sugar concentration; but it is only within the last few years, since blood sugar determination has become almost a routine clinical procedure, that enough data has been accumulated to show that Bernard was right. The blood sugar concentration in healthy fasting human beings and laboratory animals is usually within the limits 0.08 to 0.10 per cent. The average blood sugar concentration in 722 fasting diabetics appearing at Joslin's office was 0.21 per cent; in severe cases it was over 0.24 per cent, and occasionally over 0.40 per cent (218).

3. *In other diseases.* Hyperglycemia is associated with the glycosuria in cases of hypertension (247, 385, 269). There may be no relationship between the height of the blood pressure and the degree of hyperglycemia (171, 56), yet in individual cases, with fall of blood pressure the degree of hyperglycemia diminishes (181). Hyperglycemia is associated with the glycosuria in cases of hyperthyroidism (164) and carcinoma (453).

4. *From drugs.* Hyperglycemia accompanies the glycosuria due to calcium compounds (434), tartrate (437), diuretin (376, 367), ether anesthesia (103, 105), quinin (417) and morphin (378).

5. *From cooling.* The glycosuria due to cooling is accompanied by hyperglycemia (101, 247, 404, 134, 269, 231,

176 Thirty-Fifth Annual Meeting

232, 439). The glycosuria sometimes appearing in fevers is also due to hyperglycemia (189).

6. *Nervous and emotional glycosuria.* The glycosuria following stimulation of the medulla or the great splanchnic nerve is accompanied by hyperglycemia (270). Emotional glycosuria is due to hyperglycemia (183, 184, 392, 214).

7. *From epinephrin.* Soon after the discovery that epinephrin causes glycosuria it was shown that the glycosuria is accompanied by hyperglycemia (469, 301). The hyperglycemia in turn was found to be due to mobilization of liver glycogen (94, 403, 43, 3). When the liver is removed (438) or excluded from the circulation by tying off the vessels (108) or formation of an Eck fistula (302) epinephrin does not cause hyperglycemia. When the liver is made glycogen free by cold (370) hunger (370, 236) or by phosphorus poisoning (131, 132, 302) or phlorhizin ingestion (370) epinephrin does not cause hyperglycemia or glycosuria. As a matter of fact the hyperglycemia and glycosuria due to stimulation of the nervous system (273, 143, 279, 221) caffein, strychnine, and diuretin (327), quinin (417), carbon monoxide poisoning (223) and emotional disturbances are really due to adrenalin secretion and do not appear after adrenalectomy.

(c) GLYCOSURIA WITHOUT HYPERGLYCEMIA

1. *From phlorhizin.* In 1886 von Mehring (290) reported that phlorhizin, a glucoside obtained from the bark of pear and apple trees, causes glycosuria and at the same time lowers the blood sugar concentration. Von Mehring's discovery has been frequently confirmed (303, 251, 84, 355, 131, 106, 219).

2. *Renal action of phlorhizin.* One investigator (243) alleged hypertrophy and hyperplasia of the Islands of Langerhans following phlorhizin injection but this has since been disproved (423, 244). All other investigations of the

phenomenon indicate that the drug acts directly on the kidney. If the kidneys are previously removed phlorhizin does not lower the blood sugar concentration (303, 254). If the drug is injected but kept from reaching the kidneys by ligation of the renal vessels the blood sugar does not fall (251). The renal nature of the phlorhizin action is shown further by the fact that if the drug is injected into the renal vessels on one side sugar appears in the urine secreted by the kidney on that side several minutes before it appears in the urine from the other kidney (470, 349, 246). And the amount of sugar is greater in the urine secreted on the side of injection (349). The concentration of sugar in the blood of the renal vein as it leaves the kidney is appreciably less than that of the blood in the renal artery as it reaches the kidney (473). From all of which the conclusion seems warranted that phlorhizin glycosuria is not due to hyperglycemia but to increased kidney permeability. The phlorhizin produces such a change that the blood sugar which ordinarily does not pass through the kidney into the urine until its concentration has become abnormally high passes through when present at normal or even below normal concentration.

3. *Renal glycosuria.* In certain other forms of glycosuria there is no hyperglycemia. The blood sugar is normal in the glycosuria of pregnancy (130, 330) and in the glycosuria due to poisoning with sublimate (130), cantharides (247, 249, 275, 276), salt (285, 435, 436) and mushrooms (4).

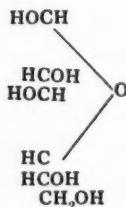
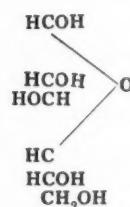
(d) NATURE AND DISTRIBUTION OF BLOOD SUGAR

1. *Distribution.* It has frequently been alleged that the blood sugar is all in the plasma. Later investigators have proved that this is not so; the glucose is fairly evenly divided between corpuscles and plasma (154, 100, 460, 166, 326, 122, 124, 210). There is some evidence that in diabetes the con-

centration in the plasma is slightly higher than in the corpuscles (122, 210).

2. *Free and combined glucose.* In 1897 Henriques raised the question of whether or not some of the blood sugar is not combined in some form that escapes ordinary analysis (179). But the studies of Michaelis and Rona (374, 375) demonstrating that all the blood glucose is dialysable show that this cannot be the case. There has, nevertheless, been a long controversy on this point and references to differences in the proportion of free and combined glucose in normal and diabetic blood still appear in the most recent literature (66).

3. *Different forms of glucose.* In 1899 Pavy declared that glucose is not the only sugar in the blood; that there is another reducing sugar whose identity is uncertain (347, 348). In 1917 Clark (77) noted that perfusion of the pancreas with Locke's solution containing dextrose causes some change in the glucose; the copper reducing value remains the same, but the optical rotation diminishes and the perfusate yields an osazone with a different melting point. He suggested that the pancreas contains an enzyme which changes the dextrose into a sugar which can be utilized by the body. In 1920 Wewith and Pryde (450) alleged that in contact with living tissue α and β glucose rapidly changes to γ glucose a compound not detectable by

 α -glucose β -glucose

Fehling's test first described by Fisher in 1914 (116), whose constitution as formulated by Irvin, Fyffe, and Hogg (194) is given as a mixture of the two substances:



In 1923 Winter and Smith (458, 459) reported that normal blood on standing shows a gradual rise in polarization value until the polarization value agrees with the reduction value for glucose. Diabetic blood shows no such change in polarization value. This they explain as due to the gradual change of the unstable γ -glucose of normal blood with its low polarization value to α - β -glucose, whereas diabetic blood, on account of the absence of the enzyme necessary for changing α - β -glucose into γ -glucose, does not contain the latter. Forrest, Smith and Winter (128) have suggested that the cause of the apparent disappearance of glucose from the blood after meals is the change of freshly absorbed α - β glucose to γ -glucose. They allege that only γ -glucose can be directly utilized by the body which explains why the α - β glucose of diabetic blood accumulates in the blood. They observed that insulin causes an alteration in the rotary power without a corresponding change in the copper reducing power which they interpret as a shifting in the equilibrium among the three forms of glucose in the direction of the normal γ -glucose the only one of the three available to the body (458). The increase in the glucose content of the blood after adrenalin injection they explain as due to an increase in the α - β form of glucose (459). All this is uncertain and still under investigation however. The most recent investigations in America (481), Germany (421) and France (313) do not confirm the English investigations.

(e) NORMAL POST-PRANDIAL HYPERGLYCEMIA

1. *Alimentary hyperglycemia.* As early as 1908, Gilbert and Badouin (146) demonstrated that there is an increase in the blood sugar concentration an hour after ingestion of glucose with a return to the normal at the end of two hours. But this post-prandial hyperglycemia was not further studied until 1913, when a micro method for blood sugar determination devised by Bang (22) made it possible for several investigators to confirm it (117, 449, 51). By determining the blood sugar concentration with this new micro method at 15 minute intervals Bang demonstrated a gradual increase in the blood sugar concentration beginning in 15 minutes and continuing for an hour, followed by a gradual decrease for two hours more. Since 1913, thousands of observations of the blood sugar concentration, after ingestion of food have confirmed the facts, observed by Bang; and we now have a pretty thorough knowledge of the influence of different amounts and kinds of food and the condition of the health on the form of the curve of post-prandial hyperglycemia.

2. *Initial rise.* Graham (155) and Sakaguchi (380) have detected a rise in the blood sugar concentration as early as ten minutes after ingestion of glucose; and Jacobsen (197) as early as five minutes.

3. *Time of maximal concentration.* The greatest number of observers have found the maximal concentration about 30 minutes after ingestion of the glucose (197, 191, 168, 405, 207, 157). Graham (155) found the maximal concentration after 20 to 30 minutes; Sakaguchi (380), after 20 to 40 minutes; Goto and Kuno (153), after 20 to 60 minutes. Hammam and Hirschman (168) have reported several cases in which the maximal concentration was reached in less than 15 minutes.

4. *Maximal concentration.* Hammam and Hirschman report the maximal blood sugar concentration after glucose ingestion 0.14 per cent (168); Hopkins, 0.156 per cent (191); Spencer, 0.135 to 0.166 per cent (405); Sakaguchi, 0.133 to 0.191 per cent (380). In a series of 53 cases Goto and Kuno (153) found that the average fasting blood sugar concentration of 0.092 per cent rose to a maximum of from 0.125 to 0.185 (an average of 0.142) per cent after glucose ingestion. In a series of about 900 cases Gray (157) found an average maximal rise to 0.14 per cent; values above 0.16 per cent were unusual. Sherril (401) has reported a rare case as high as 0.21 per cent. Jacobsen (197) has reported a rise to as high as 0.23 per cent. John (207) has reported one case as high as 0.28, another as high as 0.42 per cent.

5. *Duration.* All investigators agree that in normal cases the blood sugar concentration returns to the fasting level in from 1½ to 2 hours after glucose ingestion (202 to 213, 405, 191, 197, 167, 168, 380).

6. *Constancy of form.* Olmstead and Gay (334) have shown that the form of the blood sugar curve for any one individual examined at intervals remains constant. Jantzen of the Home Office Staff of the New England Mutual has noted a modification within normal limits of the curve of post-prandial hyperglycemia as the result of physical or mental activity; the maximal rise is not so great, it does not come so soon and the concentration remains high longer than usual (472).

7. *Examples.* Two typical blood sugar curves follow: Table 3 and the chart.

Table 3

Normal Curves of Post-prandial Hyperglycemia (197)

| Number of Minutes After Glucose Ingestion | Blood Sugar Concentration | |
|---|---------------------------|----------|
| | Case #2 | Case #11 |
| Before | 0.097 | 0.102 |
| 5 | 0.116 | 0.100 |
| 15 | 0.135 | 0.132 |
| 30 | 0.135 | 0.188 |
| 45 | — | 0.205 |
| 60 | 0.115 | 0.209 |
| 90 | 0.112 | 0.139 |
| 120 | 0.105 | 0.116 |

(f) POST-PRANDIAL HYPERGLYCEMIA IN DIABETES

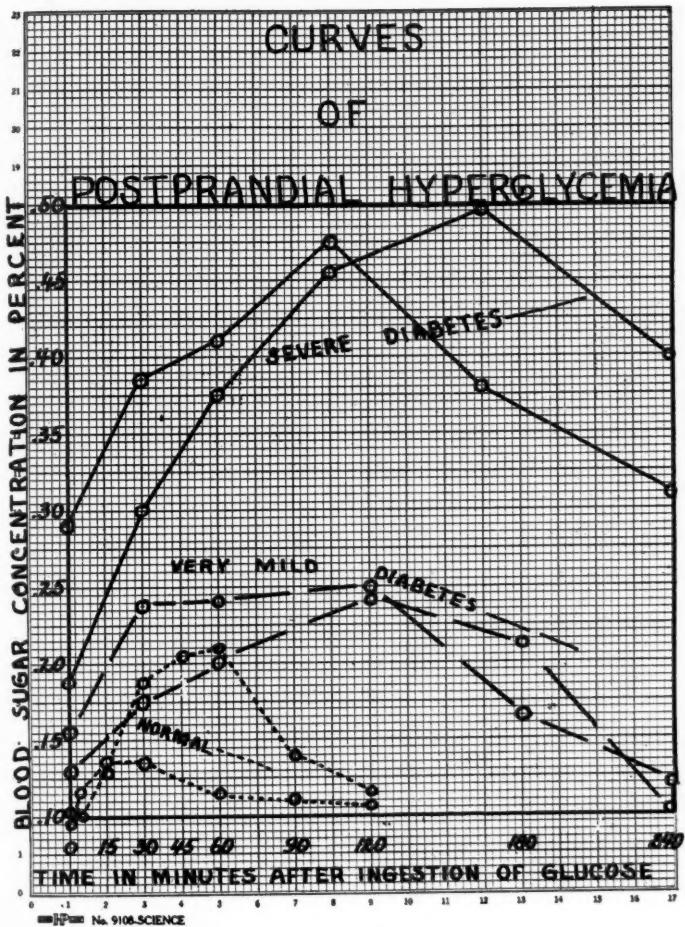
1. *The diabetic curve.* In diabetes the blood sugar concentration usually rises more slowly but reaches a higher level than in the normal. But the most constant characteristic is a delayed return to the fasting level. Instead of a return in $1\frac{1}{2}$ to 2 hours as in the normal the concentration may be still increasing at that time; the fasting level is not reached for 3 to 5 hours (193, 14, 167, 168, 405, 268, 191, 197, 157, 205 to 213).

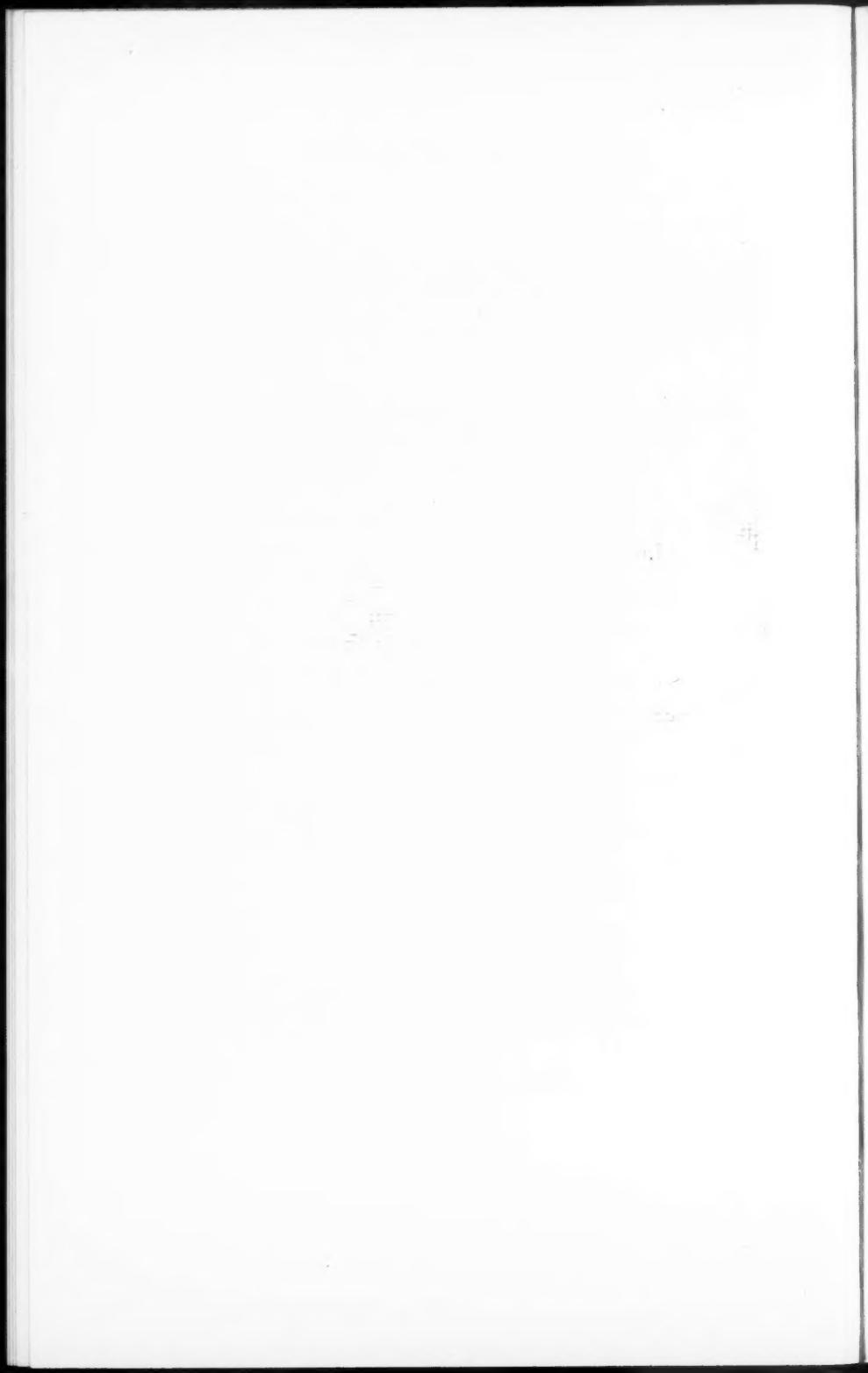
2. *Examples.* Typical diabetic curves of post-prandial hyperglycemia are shown in table 4 and the chart.

Table 4

Curves of Post-prandial Hyperglycemia in Diabetes (211)

| Number of Minutes after Glucose Ingestion | Blood Sugar Concentration | |
|---|---------------------------|-----------|
| | Case #VI | Case #VII |
| Before | 0.187 | 0.280 |
| 30 | 0.300 | 0.384 |
| 60 | 0.375 | 0.410 |
| 120 | 0.454 | 0.475 |
| 180 | 0.497 | 0.380 |
| 240 | 0.400 | 0.313 |





(g) POST-PRANDIAL HYPERGLYCEMIA IN VARIOUS CONDITIONS

1. *In other diseases.* The diabetic form of blood sugar curve after meals—delayed return to normal—has been observed in cases of hyperthyroidism (164, 167, 168, 89, 203, 204, 14, 457, 409, 311, 121, 158); hypertension and nephritis (191, 167, 168, 202, 203, 14, 238, 312, 158); gastrointestinal cancer (136, 137, 405, 121, 334); liver cirrhosis (409, 415, 158); obstructive jaundice (415, 409); thromboangiitis obliterans (158); chronic alcoholism (415); encephalitis lethargica (405); furunculosis (351, 419, 334); severe arthritis (352); myxoedema (14); and dyspituitrism (14). There is a tendency to the diabetic form of curve in individuals who are greatly fatigued (155), in menstruating women (178, 135), obesity (312, 344) and in some very old people (405). In connection with these findings in obesity and in old age it is to be noted that Jantzen (472) has noted that the maximal rise in concentration is higher on the average with the heavier and older members of a group.

2. *Effect of treatment.* In hyperthyroidism Denis, Aub and Minot (89) found the height of the blood sugar concentration after meals proportional to the severity of the disease; and these investigators as well as Wilson (457) found a change toward the normal form of curve in this disease after improvement in the condition.

3. *In endocrine disease.* In cretinism, progressive musculadystrophy, hypopituitarism, and after thyroidectomy the post-prandial rise in blood sugar concentration is only slight but the return to the fasting level is delayed suggesting that in these conditions of hypoactivity of certain endocrin glands there is an increased capacity for glucose assimilation (202, 203, 204, 160). The increased glucose assimilation in hypopituitarism is brought to the normal level again by pituitary extract (160).

(h) POST-PRANDIAL HYPERGLYCEMIA IN INCIPIENT, POTENTIAL, LATENT OR MILD DIABETES

1. *Pre-diabetic curve.* As long ago as 1914 Macleod (270) noted that the earliest sign of diabetes is a greater persistence in the post-prandial hyperglycemia; as the disease progresses this post-prandial effect persists longer and longer until finally the separate periods following meals fuse into a continuous hyperglycemia. Further study has shown that in mild diabetes there is usually a slower rise and a rise to a higher level than in the normal. But the most constant and earliest change is the characteristic diabetic delay in return to the fasting level (203, 334, 333, 208).

2. *Examples.* Table 5 and the chart show curves of post-prandial hyperglycemia in three cases of very mild diabetes.

Table 5

Post-prandial Hyperglycemia in Very Mild Diabetes (205)

| Number of Minutes After Glucose Ingestion | Blood Sugar Concentration | |
|---|---------------------------|---------|
| | Case #3 | Case #5 |
| Before | 0.156 | 0.130 |
| 30 | 0.238 | 0.176 |
| 60 | 0.240 | 0.200 |
| 120 | 0.250 | 0.243 |
| 180 | 0.165 | 0.214 |
| 240 | 0.121 | 0.100 |

(i) POST-PRANDIAL HYPERGLYCEMIA IN NERVOUS AND EMOTIONAL GLYCOSURIA

When epinephrin is given at the same time as carbohydrate food the normal post-prandial changes are reproduced but in exaggerated form (168). As might be expected from the fact that nervous and emotional glycosuria is a form of epinephrin glycosuria the curve in manic depressive insanity is likewise normal in form, but with the normal changes exaggerated (334). In a case of emotional glycosuria studied

by Folin and Berglund (124) there was a rapid rise in blood sugar concentration from 0.090 per cent to 0.189 per cent; but within two hours the concentration had fallen to 0.104 per cent. In these forms of glycosuria the delayed return to the normal level characteristic of diabetes is not found.

(k) POST-PRANDIAL HYPERGLYCEMIA IN RENAL GLYCOSURIA

1. *Incidence of renal glycosuria.* Renal glycosuria appears to have been observed first by Klemperer* who found that the blood sugar is normal and that the ingestion of carbohydrate does not increase glucose excretion. Because of the presence of albuminuria in this first case he called the condition renal glycosuria. Similar cases were reported by Lüthje (260) and Bonninger (55). Weiland (448) was the first to report a case of glycosuria with normal blood sugar but without albuminuria. Tachau (416) and Garrod (141, 142) reported similar cases. There is some question regarding the correctness of quantitative determinations of blood sugar concentration prior to 1913 and there was justification for the skepticism expressed by Allen in 1913 as to the existence of renal glycosuria (5). But reports soon showed that there is a condition with normal blood sugar concentration in which small amounts of glucose not influenced by the diet are continuously present in the urine. A great many cases of renal glycosuria have been studied in the last ten years and there can be no question that the condition exists (411, 88, 383, 257, 202, 14, 15, 240, 8, 277, 334, 224, 387, 202 to 209, 212, 213). The condition appears to be fairly common. John (208) found renal glycosuria in 99 out of 715 observations (13.8 per cent) of his cases of glycosuria. In 57 cases

*FOOTNOTE—Numerous investigators refer to Klemperer's discovery as having been reported before the Berliner Verein für innere Medizin at the meetings of May 18 and June 18, 1896, and printed in the Berliner klinischer Wochenschrift, vol. 16, 1896. But I have never been able to find reports of his paper in the copies of that volume that I have seen.

of glycosuria Gray found 9 cases of renal glycosuria (218 see p. 172). Folin and Berglund (124) state that there are usually one or two such cases out of a class of 100 students.

2. *Characteristics.* The characteristic feature of renal glycosuria is the presence of sugar in the urine without hyperglycemia. The sugar concentration of the urine is low and the total daily excretion of sugar is small. In some of the cases ingestion of glucose has no effect on the blood sugar concentration or on the quantity of sugar excreted. In other cases the curve of post-prandial hyperglycemia shows the normal slight rise and quick fall (156, 454, 343). There are other cases in which the blood sugar changes without producing a corresponding change in the sugar excretion (14, 15).

(1) THE RENAL THRESHOLD

1. *Normal.* As long ago as 1878 Claude Bernard (42) suggested that sugar appears in the urine only when the blood sugar concentration rises above a certain "threshold" value. But it was not until modern clinical micro-methods became available that it was possible to prove the existence of a threshold. Jacobsen (197), the first to determine the threshold, found an average value of 0.16 to 0.17 per cent. Other observers have found about the same value: 0.16 per cent (153, 157); 0.175 per cent (207); 0.17 to 0.18 per cent (168, 454); 0.17 to 0.20 per cent (123). In a series of 52 non-diabetics Nakayama (323) found threshold extremes of 0.08 and 0.21 per cent; but few cases were outside the limits 0.13 and 0.19 per cent; the greatest number of thresholds were between 0.14 and 0.17.

2. *High.* The threshold may be high in cases of nephritis (167, 168). In uncomplicated, mild diabetes the threshold has the normal value (453, 454, 323). If the diabetes is of

long standing (258, 453, 454, 7), or is complicated with hypertension (453, 454) the threshold may be high.

3. *Low.* The characteristic feature of renal glycosuria is the low threshold.

IV. THE PERMANENCE OF DIFFERENT TYPES OF GLYCOSURIA

1. *Types.* The facts so far justify us in recognizing five types of occasional slight glycosuria:

1. Early, incipient or mild diabetes, whether due to pancreatic disease, obesity or hypertension.
2. Glycosuria due to drugs.
3. Glycosuria due to acute infectious disease.
4. Renal glycosuria.
5. Emotional and nervous glycosuria.

2. *Drugs.* There is no reason to believe that drug glycosuria becomes diabetes; and it is highly improbable.

3. *Acute disease.* Glycosuria due to acute infectious disease is a rather heterogeneous group. There is no evidence that these cases become diabetic. Anyhow individuals with acute infectious disease are not insurable.

4. *Renal.* There is no evidence that cases of renal glycosuria become diabetic. In fact one of the characteristics of the condition emphasized by Lewis (255) is that it must remain stationary. Joslin (218) refers to one of his cases that has been under observation for 27 years (*p164*). Finlay and Rabinovitch studied one case 29 years after the onset of the symptoms and they refer to another case of 30 years' duration (112).

5. *Diabetes and shock.* It has long been believed that nervous and emotional stress and strain is an important factor in producing diabetes. The effect of emotion in producing a slight glycosuria in normal persons; the increased excretion

of sugar as the result of emotional disturbance in diabetes (324); the appearance of glycosuria with a rise in blood pressure accompanying emotional strain in cases of mild diabetes secondary to hypertension all contribute to impress us with a belief that there is some connection between emotional strain and diabetes. Further apparent testimony is afforded by the classical cases first described by von Noorden (329) and now quoted in all the text books, of the man who became diabetic as the result of surprising his wife in adultery; and of the man who became diabetic as the result of fright during a bombardment. The experimental work of Claude Bernard on the relationship between glycosuria and injury of the floor of the fourth ventricle (40) has been considered contributory evidence. One case reported by von Noorden is of peculiar interest in this connection. On a day following a terrible mental shock this man showed over one per cent sugar in the urine. Later he was able to ingest 500 grams bread a day without the appearance of glycosuria. In the 6th edition of his book von Noorden reported this man as well for ten years. Two years later in the 7th edition von Noorden reports that at the age of 52, that is, twelve years after the first appearance of glycosuria, the man developed mild glycosuria (324, p36). Such evidence might well lead to the inference that emotional glycosuria is closely related to diabetes and might give rise to diabetes.

6. *Psychosis.* We have already referred to the frequency of glycosuria in cases of nervous and mental disease. Yet diabetes is not more common among the insane than among those who are not insane (163, 407). Among 2,365 insane patients von Noorden did not find a single case of diabetes (324). As already pointed out the glycosuria occurring in depressed conditions is temporary and disappears as the condition improves. This is likewise true of the glycosuria accompanying mental diseases (391).

7. *Emotional.* Glycosuria as the result of emotional or nervous shock is very common. Haedke (165) found sugar in the urine of 15 out of 25 cases (60 per cent) after severe injuries, especially after falling a long distance with considerable physical shock, or after skull injuries. The great infrequency of diabetes following nervous or emotional strain compared with the great frequency of both strain and diabetes separately seems to prove that any apparent connection between the two in individual cases is accidental; that one does not lead to the other. Naunyn (324) was able to cite but one case in which diabetes appeared to be of nervous origin; and he speaks of how skeptical we should be in associating events as cause and effect in such cases. Cases of severe emotional and nervous glycosuria among soldiers in the great war must have been very common. Yet among the 40,000 patients who passed through Joslin's hospital center in France there were but two cases of diabetes (217). Among the many soldiers that came under Allen's observation there was not one clear-cut case of diabetes due to shock (8); there was one case with certain doubtful features about it. Von Noorden's testimony regarding diabetes among the German soldiers is to the same effect (329); the small amount of diabetes seen would have developed independently of the war. He did not see one clear-cut case of an association between shock and diabetes. In my own experience with many thousands of ex-soldiers under government care during the first four years after the war diabetes was almost non-existent.

V. HIGH MORTALITY IN THE GROUP DUE TO THE INCLUSION OF DIABETES

1. *Classification.* The evidence indicates that individuals with slight or transient glycosuria may be divided into two sub-groups which we may refer to as the diabetic and the non-diabetic groups. The diabetic group includes real pan-

190 Thirty-Fifth Annual Meeting

creatic diabetes and the milder forms of diabetes associated with obesity and arteriosclerosis; the non-diabetic group includes renal and emotional glycosuria, the glycosuria due to drugs and acute infections, and false glycosuria—cases in which the technique was at fault. It is evident from the character of the curve of post-prandial hyperglycemia in these cases that the two groups are more homogeneous with respect to carbohydrate metabolism than the original group from which they were subdivided. Are they presumably more homogeneous with respect to attributes which influence mortality?

2. *Mortality in diabetes.* Clinical experience shows that members of the diabetic sub-group do not live long. Table 6 shows how short a period elapsed from the time the disease was first recognized in a long series studied by Joslin (218) until the patient died. Even with the use of insulin the average duration of the disease among patients entering one of our best hospitals in 1923 was only 5.6 years (474).

Table 6
Duration of Life in Diabetes (218)

| Age at Onset | Number of Cases | Average Duration in Years | Number of Living Cases | Average Duration in Years |
|-----------------------|-----------------|---------------------------|------------------------|---------------------------|
| 0-10 | 33 | 2.06 | 9 | 4.44 |
| 11-20 | 48 | 2.79 | 27 | 2.70 |
| 21-30 | 40 | 3.30 | 50 | 4.40 |
| 31-40 | 53 | 4.43 | 71 | 6.12 |
| 41-50 | 71 | 6.08 | 146 | 7.04 |
| 51-60 | 97 | 6.63 | 120 | 6.29 |
| 61-70 | 52 | 6.00 | 55 | 5.38 |
| 71-80 | 14 | 3.75 | 11 | 4.45 |
| 81-90 | 0 | | | |
| Total number of cases | 408 | | 489 | |

3. *Diabetics as insurance risks.* The figures of investigators in life insurance medicine indicate that diabetes is the

hazard from which the insurance companies need to be chiefly protected in this group. In the Summer of 1906 Barringer looked up the subsequent history of eighteen individuals who had shown traces of sugar in the urine on examination for life insurance six years before and found that four of them had developed diabetes, three of them had probably developed diabetes, two of them showed a suspicion of diabetes, and nine of them were not diabetic (27). The medico-actuarial investigation (289) shows a mortality from diabetes in this group of six times the normal mortality from this cause; in the investigation of the New York Life Insurance Company (373) 32 per cent of the deaths in this group, or approximately 70 per cent of the total expected deaths were due to diabetes. The hazard from diabetes is brought out in another way: In the first five years after examination the ratio of actual to expected deaths was found to be 109; in the next five, 96; in the third five, 101; and in the fourth five, 84. In other words after the hazard from diabetes is ended—by the death of the diabetics in the first half dozen years—the hazard which remains is not so great. This very high mortality in the early years after examination was noted by Ogden in his investigation (332). The comparatively low mortality in the sub-group between the ages of 30 and 40, the ages at which emotional glycosuria is most frequent, is also in harmony with this hypothesis.

4. *Diabetic and non-diabetic glycosuria.* Since the high mortality of the group of individuals occasionally showing glycosuria is due to the inclusion of a large proportion of early diabetics the conclusion would seem to be inevitable that a simple, fraud-proof method of differentiating diabetics and non-diabetics would certainly be a very promising provisional method of dividing these cases into sub-groups which are presumably more homogeneous with respect to attributes which influence mortality.

VI. THE DIFFERENTIATION OF DIABETIC AND NON-DIABETIC GLYCOSURIA

(a) BLOOD SUGAR CONCENTRATION

The fact that some forms of glycosuria are associated with hyperglycemia and other forms with a normal blood sugar concentration may suggest quantitative blood sugar determination as a means of differentiation. But further study shows that determination of blood sugar concentration may not be of more value than determination of the urinary sugar. Under treatment or early in the morning before breakfast the blood sugar concentration of a diabetic may be normal (157). Joslin refers to a series of 48 cases of diabetes with normal blood sugar concentration when first examined.

(b) ALIMENTARY GLUCOSE TOLERANCE

1. *Glucose tolerance test.* Alimentary glucose tolerance tests have long been in use for the purpose of recognizing diabetes in suspected cases and some form of alimentary glucose tolerance test is, or has been used by several insurance companies (445, 341, 86, 118, 107, 385). Joslin has suggested examining the urine after two heavy carbohydrate meals (216).

2. *Early work.* In 1884 the Norwegian physiologist Worm-Müller (466) alleged that each animal has a definite tolerance for carbohydrate; and that any excess above the tolerance limit will be excreted in the urine. According to this investigator the ingestion of 50 grams of glucose will just suffice to produce glycosuria in a normal man. He alleged further that the tolerance is diminished in early or mild diabetes and suggested using the alimentary tolerance test as a means of diagnosing early diabetes (467). This work of Worm-Müller was repeated and extended by Hofmeister (186), Moritz (310), Block (46), Linosier (259) and Donath

and Schlesinger (92) with results that tended to confirm those of Worm-Müller. The results of Comessatti (78), Grober (161), and Hohlweg (187) indicated that exercise increased the capacity for utilizing glucose. The figures of Grober (161) and Hohlweg and Voit (188) showed an increased glucose oxidizing capacity following a rise in the external temperature. The experiments of Goetsch, Creslig and Jacobsen (149) indicated an increased glucose oxidizing capacity following operations which produced an insufficiency of the posterior lobe of the hypophysis. A diminished limit of assimilation, on the other hand, was noted by Geelvink (144), Van Ordt (337) and Raimann (360) in various neuroses. The diminution appeared to be especially marked in cases of melancholia (12, 361) and following traumatic neuroses (199, 200, 407). Haedke (165) reported a lowered glucose tolerance after severe injuries even in the absence of any neurosis. A diminished tolerance was reported also in acute alcoholism (407), in lead poisoning (407) and in Graves' disease (76, 234).

3. *Hyperglycemia and glycosuria.* In 1899 Raphael pointed out that although most diabetics, even in the early stages showed alimentary glycosuria, many normal persons show a temporary decrease in the glucose tolerance (364). In 1913 Jacobsen (197) observed glycosuria after glucose injection in 8 out of 18 normal persons. Taylor and Hulton (418) called attention to the great individual differences in glucose tolerance and pointed out that many persons can take even 400 grams of glucose without showing glycosuria. Holst (190) tested the urine of 14 healthy people and of 145 people with different diseases (none of them diabetic, however) and found that after a carbohydrate meal 31 showed glycosuria. After administration of 100 grams glucose Gray (157) found glycosuria in 40 per cent of 129 cases. Conversely, Rigler and Ulrich (369) observed a rise

in the blood sugar concentration after glucose administration to as high as 0.2 per cent without any glycosuria. John has reported numerous cases with high blood sugar following a glucose test meal and cases of diabetes with blood sugar concentration as high as 0.2 per cent without glycosuria; and conversely, cases with normal blood sugar and glycosuria (202 to 213). Case #VI (see table 4), a very severe diabetic showed no glycosuria with a blood sugar of 0.187 per cent; and showed only 0.16 per cent glucose in the urine, an amount that would be missed by the usual clinical tests, one hour after ingestion of 100 grams glucose when the blood sugar concentration was 0.375 per cent. In a series of 714 observations 13.8 per cent showed glycosuria with normal blood sugar; and 18.3 per cent showed no glycosuria in the presence of hyperglycemia (208). If a diagnosis had been based on urine examination alone the possibility of error was 32 per cent.

4. *Rate of absorption.* As long ago as 1898 Gilbert and Carnot (147) observed that the presence or absence of glycosuria after glucose ingestion depends somewhat on the rate of administration or absorption of the glucose. And this was confirmed by Doyon and Dufourt (93) and by Blumenthal (49), but the most thorough studies of the effect of rate of ingestion on glucose assimilation were carried out by Woodyatt (464, 465, 482), who devised a method for continuous intravenous glucose injection at a measured rate. Woodyatt showed that large quantities of glucose can be injected without leading to glycosuria if the rate is kept down to 0.85 grams per kilo body weight per hour. As soon as the rate of injection rises above this figure sugar appears in the urine.

5. *Renal threshold studies.* It is evident that an alimentary glucose tolerance test cannot be used to identify cases of diabetic glycosuria. The reason for this will be evident

from a study of the renal threshold. Glucose appears in the urine when the blood sugar concentration exceeds the threshold value. But blood for analysis is collected at intervals; the urine, though voided at intervals, represents a continuous secretion. An individual may, therefore, void urine containing sugar at the same moment that the blood sugar concentration is below the threshold, the sugar having been secreted shortly before when the blood sugar concentration had been above the threshold. And vice versa, an individual may void a normal urine at the same moment that the blood sugar is above the threshold, this urine having been secreted shortly before the blood sugar concentration had risen to the value found at the time of voiding. Examples of blood and urine analyses bringing out this point will be found in the literature (213). Furthermore it appears that once glycosuria has started as a result of a blood sugar concentration in excess of the threshold, the glycosuria may persist for a time after the blood sugar concentration has fallen below the threshold; in other words the occurrence of glycosuria temporarily lowers the threshold (328, 15). This would seem to be an example of a very fundamental physiological phenomenon that has been called functional inertia (471), analogous to the delay before the heart rate begins to retard after stimulation of the vagus and the corresponding delay in resuming normal rhythm for a time after the stimulation has ceased. Nor does the total amount of sugar excreted after glucose ingestion serve to identify diabetics. In many cases of even severe diabetes showing a fasting hyperglycemia the total quantity of sugar excreted after ingestion of a dose of even 100 grams of glucose may be very small (202).

196 Thirty-Fifth Annual Meeting

(c) MISCELLANEOUS METHODS

Some companies have based selection on the amount of sugar excreted, others on the ability to pass several normal specimens. Thus Balch (18) says that the Metropolitan considers 0.2 per cent sugar suspicious in a urine of low specific gravity but does not consider this amount suspicious in a urine of high specific gravity. According to Pauli the Union Central considers 0.25 per cent as the dividing line (341). The Equitable according to Wolf requires an alimentary glucose tolerance test if more than 0.25 per cent glucose is found in the urine (462). The Metropolitan emphasizes the importance of home office examinations (19) and accurate determination of the quantity of sugar in the urine (475). Daley reports that his company requires three normal specimens preferably six days apart (86). And Fisher (118) requires at least five recent samples of normal urine voided at different times of day. When mere traces are found on a first examination, Pauli requires additional samples (341). Since, with the exception of the method of over-selection referred to in the introduction, all these methods of selection are based on the results of urine analysis, and, as we have seen, the different types of glycosuria may be indistinguishable by urine analysis, I have not gone fully into the details of the methods of selection referred to above or tried to describe the methods of selection of all the companies or tried to give the latest modifications of these methods. The alimentary glucose tolerance is still in use (476, 479). Companies doing sub-standard business get around the difficulty by "rating up" applicants showing glycosuria. For those who confine themselves to urine analysis the statement of Joslin in his address before the Association of Life Insurance Medical Directors in 1921 still holds (216, p87):

" * * * it is safe to consider any patient to have diabetes mellitus * * * who has sugar in the urine demonstrable by any of the common tests."

(d) POST-PRANDIAL HYPERGLYCEMIA

1. *Types of curves.* There are three types of curves of post-prandial hyperglycemia:

The normal curve which shows a rapid rise and then a rapid return to the fasting level.

The diabetic curve which shows a rise followed by a very slow return to the fasting level.

The curve of renal glycosuria which shows little or no rise.

2. *Maximal concentration.* The maximal post-prandial value is very variable and probably depends in part on the rate of absorption (207). Furthermore, unless practically continuous determination of blood sugar concentration is made we cannot be certain that we have determined the maximal value. This maximal concentration is not a value of great practical importance.

3. *Percentile increase.* Badouin (28) suggested the term "co-efficient glycémique" for the quotient obtained by dividing the blood sugar concentration one hour after glucose ingestion by the fasting blood sugar concentration; according to Badouin the normal co-efficient is 1.35. Badouin's observation as to the value of this co-efficient and his opinion as to its significance have not been confirmed; this co-efficient has no great significance.

4. *Time required to reach maximum.* The exact time which elapses after administration of glucose before the maximal value for blood sugar concentration is reached is a variable quantity of apparently not enough significance to warrant the impractical, practically continuous analyses necessary for determining this figure.

5. *Threshold value.* A determination of the "renal threshold" serves to distinguish cases of renal glycosuria. But it

is difficult to determine and does not serve to distinguish diabetic glycosuria from emotional glycosuria.

6. *Time of return to normal.* The one characteristic which seems to be of practical importance in distinguishing the diabetic curve from the other curves is the time of the return to the fasting value. In all except the diabetic cases the blood sugar concentration two hours after ingestion of glucose has returned to the normal fasting value. In cases of diabetes of even the mildest form the blood sugar concentration two hours after ingestion of glucose is still above the normal.

(e) DETAILS OF THE BLOOD SUGAR TOLERANCE TEST

1. *Amount.* In the first blood sugar tolerance test—proposed by Janney and Isaacson (202, 203)—the blood sugar concentration was determined at half hour intervals after administration to the fasting subject of $1\frac{3}{4}$ grams glucose per kilo body weight in 40 per cent aqueous solution (334). Estimating about 66 per cent absorption and a rate of assimilation of glucose by the tissues of 0.85 gram per kilo body weight per hour (Woodyatt's figures, 464, 465) Olmstead

100
and Gay administered $(2 \times 0.85 \times \frac{100}{66}) = 2.5$ grams per

kilo body weight for a two hour test (334). But further study has shown that the character of the post-prandial blood sugar curve does not depend on the relationship of the amount of glucose ingested to the body weight. The literature on post-prandial hyperglycemia after varying quantities of glucose and other sugars and after protein meals and mixed diets show that there are some differences in the form of curve depending on the nature and quantity of the food given; but the same type of curve is obtained after any foodstuff which can serve as a source of glucose (198, 129, 38, 124). The

rise in blood sugar concentration may be as great after 30 grams glucose as after 200 grams (38); and as little as even 10 or 20 grams glucose may give rise to post-prandial hyperglycemia (406). The return to normal is more rapid with these smaller quantities however. Body weight does not appear to influence the form of the curve obtained after ingestion of 100 grams glucose (397). In by far the largest proportion of studies reported in the literature—300 out of 479 in a series looked up by Gray (157)—100 grams glucose has been the dose; in the case of this dose we have, therefore, the largest number of controls for comparison. 100 grams of glucose represents 400 calories which is considerably in excess of the caloric needs of the body for a period of two hours; this amount would, accordingly, seem to be a good test of the storage capacity for carbohydrate.

2. *Form of administration.* In attempts to control more precisely the rate at which glucose enters the blood intravenous administration has been tried (422, 424, 369); but the results have not proved more valuable than those obtained through oral administration. As a matter of fact the rate of intestinal absorption appears to be very uniform (292-294, 296-298, 203, 334). Furthermore the rate of absorption is not very important (117). Experiments suggest that the comparatively small quantity of glucose first absorbed is responsible for the hyperglycemia and that the glucose later absorbed is rapidly assimilated without increasing the blood sugar concentration; a considerable amount of the sugar administered may sometimes be found in the stomach an hour after it has been administered (31).

3. *Criteria for selection.* A survey of all the available curves of post-prandial hyperglycemia shows that in non-diabetics the return to a blood sugar concentration below 0.12 per cent is reached within two hours after glucose ingestion. In diabetes of even the mildest grade the blood sugar con-

centration is still above 0.12 two hours after glucose ingestion.

(f) OUR METHOD OF SELECTION

On the basis of the facts outlined in the preceding paragraphs we have devised the following test for dividing insurance applicants without other impairment than slight glycosuria on one or two occasions into an acceptable and an unacceptable group: The applicant swallows 100 grams glucose dissolved in 250 cc. of cold water. Two hours later the sugar concentration is determined by the method of Folin and Wu (127) in blood drawn from the median cephalic vein. Applicants showing less than 0.12 per cent blood sugar are accepted; those showing more than 0.12 per cent are not accepted.

VII. CRITICAL CONSIDERATIONS

(1) *Sub-standard insurance vs. medical selection.* This paper deals only with the medical selection system of dealing with the glycosuria problem and not at all with the sub-standard insurance system. The comparative merits of the two systems lie entirely outside the scope of the paper. It would seem, however, that where the two systems come into competition the disastrous experience of the British government in dealing with a heterogeneous group would be repeated. In 1690 annuities were issued at a price based on the assumption that the average expectation of life of the population as a whole, *i. e.*, without respect to age, was seven years, a correct but naive assumption that did not take into account the effect of selection against the government—young people bought, old people did not. Suppose now a heterogeneous group with a mortality of 120 per cent of the standard can be divided in two groups equal in number one with a mortality of 60 and the other with a mortality of 180. And suppose the "good" members of the group take advantage of medical selection and

low rates and only the "bad" members with a mortality of 180 are left to take substandard insurance at the assumed rate of 120!

2. *Single tests. Theory.* Objection may be raised to our assumption that members of a group can be identified by single tests. This objection is based on a failure to distinguish the two conceptions: life duration of an individual, and mortality of a group. Factors which control life duration may be divided into two categories:

(a) Certain determinate attributes such as age, sex, height, weight, etc.,

(b) Certain indeterminate accidentals such as casualties from conflagration, murder, automobiles, etc.

The determinate attributes have a relatively slight but wide spread influence* on all lives in a group. The influence of the indeterminate accidentals is concentrated on a relatively small proportion of the total lives in any group; on these lives this influence may wholly control life duration. By eliminating to some extent applicants especially exposed to casualties the insurance companies select in such a way that the total effect of indeterminate accidentals is less than the total effect of the determinate attributes. No amount of experience will enable us to predict the duration of individual lives because the influence of no single attribute on mortality is sufficiently potent to overcome the possible effect of the indeterminate accidentals. But the proportion of casualties in any group is so small that if individuals are divided into groups homogeneous with respect to attributes influencing mortality, weight of numbers overcomes the effect of accidentals. Briefly stated: indeterminate accidentals may control the life duration of an individual; certain determinate attributes control the mortality of a group. No significance need then be attached to the unusual life duration in individual cases; in

202 Thirty-Fifth Annual Meeting

this respect occasional errors in identification come in the same category as occasional automobile accidents.

3. *Single tests. Practice.* Indeed we must base our decisions on the results of single simple tests; we cannot send life insurance applicants to a hospital for an elaborate examination and repeated tests. And twenty years of practical experience has justified the assumption that we can safely make single tests the basis of selection. After an exhaustive study of the subject of albuminuria and casts Dwight proposed in 1905 (96) that if an applicant with no impairment except a history of albuminuria or casts could at some time pass a specimen of urine that is normal in every respect he should be considered insurable at standard rates. From the point of view of the clinician this may seem an arbitrary decision but the crucial test of actual experience has demonstrated that this group of individuals has an acceptable mortality. In a series of 4,905 exposures in this group there were only 17 deaths instead of the expected 35.5 (44). As a life insurance medical problem the question is not primarily that of the relationship of albuminuria to nephritis; few of those with albuminuria died of nephritis (44, 480). From the point of view of life insurance medical science the essential facts are that the group of individuals who have at some time shown albumin or casts in the urine has an excessive mortality. But this group is not homogeneous; it may be divided into two more homogeneous sub-groups:

- (a) Those sometimes able to pass a normal urine,
- (b) Those never able to pass a normal urine.

Actual experience has shown that the mortality of group "a" is within acceptable limits.

4. *Determination of morality.* It may be objected that although division of individuals showing occasional slight glycosuria into two sub-groups, a diabetic and a non-diabetic

group and the identification of the members of the two sub-groups, solves the clinical problem of the recognition of diabetes it may not solve our insurance problem of selecting goods risks among these same individuals; and that this is a return to the old unscientific guessing principle of selection on the basis of attributes whose effect on mortality we do not actually know. The two problems mentioned here are, indeed, not necessarily identical; we recognize that the insurance problem requires a method of identifying individuals as members of a sub-group whose mortality is known to be acceptable. But this method of selection is not a return to the old guessing method. We accept completely the modern scientific principles of life insurance medical science according to which selection should be based upon grouping with respect to attributes whose influence on mortality has been determined by experience. But we cannot accept the characteristics recorded on our old records as final in their completeness and continue to reject groups which have been found below standard simply because none of the recorded characteristics influence the mortality of these groups sufficiently to be of use as a basis of further subdivision. From time to time we must add new attributes for subdividing borderline groups thereby devising provisional methods of selection for the purpose of determining mortality by actual experience. Life insurance medicine should be recognized and cultivated as a science independent of clinical medicine with methods, problems, and a point of view of its own. The determination of the influence of various factors on mortality is the most legitimate field of research in this science.

5. *Accepted diabetics.* The criticism may be offered that, although this test enables us to recognize early or mild diabetes in those in whom the test is carried out, it does not enable us to detect early or mild diabetes in those who, happening to pass a normal urine on their first insurance

204 Thirty-Fifth Annual Meeting

examination and not being submitted, therefore, to the test are accepted as without impairment. But it is to be noted that our present problem is only that of subdividing the heterogeneous group of *bad* risks with a high mortality—the group showing glycosuria at some time—into two more homogeneous sub-groups one of which is good. Our present problem is not that of subdividing the heterogeneous group of *good* risks who show no recognizable impairment into a good group and a bad group; for this great group—which includes the unrecognized mild diabetics—has a mortality of only 50 to 60 per cent of the expected. In theory we ought to recognize these cases; in practice we do not have to. The immediate problem is not that of rejecting more but of accepting more.

6. *Fraud.* Deliberate fraud does not give the insurance company much trouble. We issue insurance only to individuals with a good reputation, and most people with a good reputation have a good character. The few who get insurance dishonestly come into the large group of persons with no discoverable impairment; a mortality of only 50 to 60 per cent of the standard in this group indicates that the number of frauds is not great enough to make the problem a pressing one. The advent of insulin does not increase the probability of fraud. The amount of insulin required to metabolize 100 grams of glucose in a diabetic would be so large that unless just the right amount were given at just the right time the danger of a hypoglycemic reaction from an overdose would be very great. A dishonest applicant would need to know exactly how and when our test was to be carried out; then with the aid of a physician who could make blood sugar determinations he would have to make repeated tests, taking increasing amounts of glucose with increasing doses of insulin until he had worked up to 100 grams glucose with the dose of insulin which would suffice in his case to take

care of just this amount of glucose without causing a dangerous hypoglycemic reaction. In addition, he would have to experiment with the time relations of the insulin action in his case. And his efforts might even then be in vain for there is no constancy in the quantitative effect of insulin (123, 359, 264, 397, 222). The difficulty, danger and expense of such an undertaking would seem to be considerable enough to make it impractical for any but the most deliberate fraud and such a rascal would be astute enough to avoid the necessity of the test by using dietetic measures to obtain a sugar-free urine in the first place.

7. *Functional efficiency tests.* Our company has now made a single test of functional efficiency under load the basis of selection in the three largest groups of doubtful cases, the borderline circulatory cases, the borderline albuminuria cases, and the borderline glycosuria cases. To appreciate the significance of this fact we have only to recall what a large proportion of insurance applicants show one of these impairments.

1. Abeles, M.: Ueber ein Verfahren zum Enteiweiessen des Blutes fur die Zuckerbestimmung.—Zeitschr fur Physiol. Chem. 15, 495, 1891.
2. Addis, T., and Shevky, A.: A Modification of the Picrate Method for Blood Sugar Determinations.—Jour. of Biol. Chem. 35, 53, 1918.
3. Agadschanianz, K.: Ueber den Einfluss des Adrenalin auf des Leber und Muskeln enthaltende Glykogens.—Biochem. Zeitschr. 2, 148, 1907.
4. Alexander, M.: The Occurrence of Glycosuria in Mushroom-Poisoning with the Report of 5 Cases in Which a Mild Nephritis and a Renal Glycosuria were the Persistent and Predominating Features.—Amer. Jour. of Med. Sciences 159, 543, 1920.
5. Allen, F.: Glycosuria and Diabetes.—Cambridge, 1913.
6. Allen, F.: Diabetes Mellitus.—Nelson's Loose Leaf Medicine. 3, 55, 1920.
7. Allen, F., and Wishart, M.: Experiments on Carbohydrate Metabolism and Diabetes. II. The Renal Threshold for Sugar and Some Factors Determining it.—Jour. of Biol. Chem. 43, 129, 1920.
8. Allen, F., Wishart, M., and Smith L.: Three cases of "Renal Glycosuria."—Arch. of Int. Med. 24, 523, 1919.

9. Anders, J., and Jameson, H.: "The Relation of Glycosuria to Pituitary Disease, with Statistics."—Am. Jour. Med. Sci., 1914, cxlviii, 323.
10. Araki, T.: Ueber die Bildung von Milchsaure und Glucose im Organismus bei Sauerstoffmangel.—Zeitschr. für physiol. Chem. 16, 453, 1892.
11. Araki, T.: Beiträge zur Kenntnis der Einwirkung von Phosphor und von arsenige Saure auf den thierischen Organismus.—Zeitschr. für physiol. Chem. 17, 311, 1893.
12. Arndt.: Ueber alimentare Glykosurie bei einiger Neurosyphosis.—Berl. klin. Wochenschr. 35, 1085, 1898.
13. Autenrieth, W., and Tesdorff, T.: Ueber eine kolorimetrische Bestimmung des Traubenzuckers im Harn.—Münch. med. Wochenschr. 57, 1780, 1910.
14. Bailey, C.: Studies on Alimentary Hyperglycemia and Glycosuria.—Arch. of Int. Med. 23, 455, 1919.
15. Bailey, C.: Renal Diabetes.—Amer. Jour. of Med. Sciences, 157, 221, 1919.
16. Baisch, K.: Ueber die Natur der Kohlenhydrate des normalen Harnes.—Zeitschr für physiol. Chem. 19, 339, 1894.
17. Baisch, K.: Nachtrag zu der Mitteilung "Ueber die Natur der Kohlenhydrate des normalen Harnes."—Zeitschr für physiol. Chem. 20, 248, 1895.
18. Balch in discussion of paper by Wolf: A Study of the More Common Methods Used to Detect Sugar in Urine, with Special Reference to the Tests Employed in the Laboratory of the Equitable Life Assurance Society.—Trans. of the 31st annual meeting of the Association of Life Insurance Medical Directors, 1920, 333.
19. Balch.: Discussion of Joslin's paper.—1921 meeting of the Assoc. of Life Ins. Med. Dir.
20. Bang, I.: Zur Methodik der Zuckerbestimmung.—Biochem. Zeitschr. 2, 271, 1906-07.
21. Bang, I., and Bohmansson, G.: Zur Methodik der Harnzuckerbestimmung.—Zeitschr für physiol. Chem. 63, 443, 1909.
22. Bang, I.: Ein Verfahren zur Mikrobestimmung von Blutbe standteilen.—Biochem. Zeitschr. 49, 19, 1913.
23. Bang, I.: Der Blutzucker, 1913.
24. Bang, I., Lyttkens, H., and Sandgren, J.: Ueber die Bestimmung des Blutzucker.—Zeitschr für physiol. Chem. 64, 497, 1910.
25. Barringer, T.: The Incidence of Glycosuria and Diabetes in New York City Between 1902 and 1907 With a Report of Two Cases of Essential Pentosuria.—Arch. of Int. Med. 3, 295, 1909.
26. Barringer, T.: The Effect of Exercise Upon the Carbohydrate Tolerance in Diabetes.—Amer. Jour. of Med. Sci. 151, 181, 1916.
27. Barringer, T., and Ropes, J.: The Prognosis of Transient Spontaneous Glycosuria and its Relation to Alimentary Glycosuria.—American Journal of Medical Science 133, 842, 1907.
28. Baudouin, A.: Etude sur quelques glycémie.—La glycémie expérimentales. Paris, 1908.
29. Baumann, E., and Isaacson, R.: An Adaptation of the Folin and Wu Blood Sugar Method Applicable in Small Amounts of Blood.—Jour. of Lab. and Clin. Med. 7, 357, 1921.

30. Beard, A., and Grave, F.: Renal Glycosuria.—Arch. of Int. Med. 21, 705, 1918.
31. Beeler, C., Bryant, A., Cathcart, E., and Fitz, R.: An Improved Alimentary Glucose Tolerance Test.—J. Metab. Res. 1, §549, 1922.
32. Benedict, S.: A Modification of the Lewis-Benedict Method for the Determination of Sugar in the Urine.—Jour. of Biol. Chem. 34, 203, 1918.
33. Benedict, S.: Note on the Determination of Blood Sugar by the Modified Picric Acid Method.—Jour. of Biol. Chem. 37, 503, 1919.
34. Benedict, S., and Osterberg, E.: A Method for the Determination of Sugar in Normal Urine.—Jour. of Biol. Chem. 34, 195, 1918.
35. Benedict, S., and Osterberg, E.: Studies in Carbohydrate Metabolism. I. A Preliminary Report on the Sugar Eliminated in the Urine of the Normal Dog.—Jour. of Biol. Chem. 34, 209, 1918.
36. Benedict, S., Osterberg, E., and Neuwerth, I.: Studies in Carbohydrate Metabolism. II. A Study of the Urinary Sugar Excretion in Two Normal Men.—Jour. of Biol. Chem. 34, 217, 1918.
37. Benedict, S., and Osterberg, E.: A Method for the Determination of Sugar in Normal Urine.—Jour. of Biol. Chem. 48, 51, 1921.
38. Bergmark: Zuckersorption und Blutzuckerspiegel.—Jahrb. für Kinderheilk. N. F. 80, 373, 1914.
39. Bernard, C.: De l'origine du sucre dans l'économie animale.—Arch. gén. de Méd. 4 Sér. vol. 18, 303, 1848.
40. Bernard, C.: De l'origine du sucre dans l'économie animale.—Mém. de la soc. de biol. Paris 1, 121, 1849.
41. Bernard, C.: Note sur la présence du sucre dans le sang de la veine porte et dans le sang des veines hépatiques.—Compt. rend. de l'acad des science Paris, 40, April 2, 1855.
42. Bernard, C.: Lecons sur le diabète.—Paris, 1878.
43. Bierry, H., et Gatin—Gruzewski, Z.: Action physiologique de l'adrénaline pure—Comptes rend. de la soc. de biol. 58, 902, 1905.
44. Blakely, D.: Urinalysis: A Review of Ten Years Experience of the New England Mutual.—Proceed. of the 27th Annual Meet. of the Assoc. of Life Ins. Med. Directors, 1916, 419.
45. Blakely in discussion of a paper by Patton: The Importance of Glycosuria in Life Insurance. Transactions of the 27th annual meeting of the Assoc. of Life Ins. Med. Directors, 1916, 479.
46. Bloch, G.: Ueber alimentare Glykosurie.—Zeitschr für klin. Med. 22, 525, 1893.
47. Blum, F.: Ueber Nebennierendiabetes.—Deutsch Arch. für klin. Med. 71, 146, 1901.
48. Blum, F.: Weitere Mitteilungen zur Lehre von dem Nebennierendiabetes.—Arch. für die gesamte Physiol. 90, 617, 1902.
49. Blumenthal, F.: Zur Lehre von der Assimilationsgrenze der Zuckerarten.—Beiträge zur chem. Phys. und Path. 6, 329, 1905.
50. Bock, C., und Hoffman, F.: Ueber eine neue Entstehungsweise

von Melliturie.—Arch für Anat. Physiol. und wissenschaftl. Med. 1871, 550.

51. Böe: Untersuchungen über alimentare Hyperglykämie.—Biochem. Zeitschr. 58, 106, 1913-14.
52. Böhm, R., und Hoffman, F.: Beiträge zur Kenntniss des Kohlenhydratstoffwechsels.—Arch. für exp. Path. und Pharmak. 8, 271, 1877-8.
53. Bohmansson, G.: Über den qualitativen Nachweis des Harnzuckers.—Biochem. Zeitschr. 19, 281, 1909.
54. Bond, C.: The Relation of Diabetes to Insanity.—Brit. Med. Jour. 1895, 777.
55. Bonninger, M.: Beitrag zur Frage des Nierendiabetes.—Deutsch. med. Wochenschr. 34, 780, 1908.
56. Botti, A.: Lo zuechero del sangue negli arteriosclerotici ipertensive II. Policlinico 29. Sezione Pratica 249, 1922.
57. Breul, L.: Kann der Zuckergehalt des normalen Harnes durch einseitige Ernährungsweise und andere noch in den Bereich des physiologischen fallende Bedingungen zu höheren Graden gesteigert werden.—Arch. für exp. Path. und Pharmak. 40, 1, 1898.
58. Broeslauer, D., und Sterke, H.: Der Einfluss von Muskelarbeit auf den Blutzuckergehalt.—Deutsch Arch. für klin. Med. 130, 358, 1919.
59. Brücke, E.: Über das Vorkommen von Zucker in Urin Gesunden Menschen.—Sitzungsber der kon-kais. Akad. der Wissensch. zu Wien. 1858: Math-Natur. Klasse. 29, 346.
60. Brücke, E.: Ueber die Glykosurie der Wöchnerinnen.—Wien. Med. Wochenschr. 8, 319 and 337, 1858.
61. Brücke, E.: Darf man Urin, in welchem der Zucker quantitativ bestimmt werden soll, vorher mit Bleiessig ausfallen? —Untersuchungen zur Naturlehre des Menschen und den Thiere (Moleschott's Untersuchungen) Giessen 7, 70, 1860.
62. Brücke, E.: Darf man Urin in welchem der Zucker quantitativ bestimmt werden soll, vorhr mit Bleiessig ausfallen? —Sitzungsber. der kon-kais. Akad. der Wissensch. zu Wien. 39, 10, 1860.
63. Buhl: Mitteilungen aus der Pfeuffer'schen Klinik. Epidemische Cholera.—Zeitschr für rationelle Med. N. F. 6, 1, 1855.
64. Bumm, E.: Ueber transitorische Albuminurie und Melliturie beim Delirium.—Berl. klin. Wochenschr. 19, 378, 1882.
65. Cammidge, P.: Glycosuria and Allied Conditions.—N. Y. 1913.
66. Cammidge, P.: The Insulin Treatment of Diabetes Mellitus.—New York 1924.
67. Cammidge, J., Forsyth, J., and Howard, H.: Some Factors Controlling the Normal Sugar Content of the Blood.—Brit. Med. Jour. 2, 586, 1921.
68. Cannon, W.: The Emergency Function of the Adrenal Medulla in Pain and the Major Emotions.—Amer. Jour. of Physiol. 33, 356, 1914.
69. Cannon, W., Shohle, A., and Wright, W.: Emotional Glycosuria.—Amer. Jour. of Physiol. 29, 280, 1911.
70. Carlson, A., and Drennan, F.: The Control of Pancreatic Diabetes in Pregnancy by the Passage of the Internal Secretion of the Pancreas of the Fetus to the Blood of the Mother.—Amer. Jour. of Physiol. 28, 391, 1911.
71. Carlson, A., Orr, J., and Jones, W.: The Absence of Sugar

in the Urine After Pancreatectomy in Pregnant Bitches Near Term.—*Jour. of Biol. Chem.* 17, 19, 1914.

72. Castellani, A., and Willemore, J.: Glycosuria of Malarial Origin.—*Brit. Med. Jour.* 1921, 2, 286.
73. Cawley, T.: A Singular Case of Diabetes Consisting Entirely in the Quality of the Urine; with an Inquiry into the Different Theories of that Disease.—*The London Medical Journal*, 9, 286, 1788.
74. Chauveau, M., et Kaufman, M.: Consequences physiologiques de la détermination de l'activité spécifique des échanges ou du coefficient de l'activité nutritive et respirative dans les muscles en repos et en travail.—*Compt. rend. des séances de l'académie.* 104, 1352, and 1763, 1887.
75. Chevreul: Sur le sucre de diabète.—*Annales de chimie.* Paris, 95, 319, 1815.
76. Chvostek, F.: Ueber alimentare Glykosurie bei Morbus Basedowii.—*Wien klin. Wochenschr.* 5, 251, 267 and 325, 1892.
77. Clark, A.: The Interrelation of the Surviving Heart and Pancreas of the Dog in Sugar Metabolism.—*Jour. of Exp. Med.* 26, 731, 1917.
78. Comessatti, G.: Ueber die Aenderung der Assimilationsgrenze für Zucker durch Muskelarbeit.—*Beiträge zur chem. Physiol. und Path.* 9, 67, 1907.
79. Couré, D., and Parsons, J.: Studies on Blood Sugar. Effect of Blood Constituents on Picrate Solutions.—*Arch. of Int. Med.* 26, 333, 1920.
80. Csanka, F.: The Influence of Ingested Carbohydrate, Protein and Fat on the Blood Sugar in Phlorhiz in Diabetes.—*Jour. of Biol. Chem.* 26, 93, 1916.
81. Csanka, F., and Taggart, G.: Note on the Reliability of the Benedict and Folin-Wu Blood Sugar Determination.—*Jour. of Biol. Chem.* 54, 1, 1922.
82. Cullen, W.: First Lines of the Practice of Physic.—*Edinburgh 1791.*
83. Cummings, R., and Piness, S.: A Study of Blood Sugar. A Comparison of the Tolerance for Glucose in Diabetic and Normal Subjects.—*Arch. of Int. Med.* 19, 777, 1917.
84. Von Czyhlarz, E., und Schlesinger, W.: Blutzuckeruntersuchungen bei Phlorhizindiabetes.—*Wien. Klin. Rundschau.* 15, 743, 1901.
85. Dakin, H., and Dudley, H.: The Fate of 1-Alanine in the Glycosuric Organism.—*Jour. of Biol. Chem.* 17, 451, 1914.
86. Daley, W., in discussion of paper by Patton: The Importance of Glycosuria in Life Insurance.—Transactions of the 27th annual meeting of the Assoc. of Life Ins. Med. Directors, 1916, 483.
87. Dawson, W.: Glycosuria and Insanity.—*Medical Press and Circular.* 1, 73, 1902.
88. De Langen, C.: Beitrag zur Kasuistik des renalen Diabetes.—*Berl. klin. Wochenschr.* 51, 1792, 1914.
89. Denis, W., Aub, J., and Minot, A.: Blood Sugar in Hyperthyroidism.—*Arch. of Int. Med.* 20, 964, 1917.
90. DeWesselow, O.: The Picric Acid Method for the Estimation of Sugar in Blood with a Comparison of This Method with That of Maclean.—*Biochem. Jour.* 13, 148, 1919.
91. Dobson, M.: Experiments and Observations on the Urine in

210 Thirty-Fifth Annual Meeting

a Diabetic.—Medical Observations by a Society of Physicians in London. 5, 1776.

92. Donath, J., und Schlesinger, W.: Blutzuckerbestimmungen bei alimentarer Glykosurie beim Hunde.—Wien. klin. Rundschau. 15, 749, 1901.
93. Doyon, M., et Dufourt, E.: Sur les conditions expérimentales de la consommation tissulaire du glucose injecté dans les veines.—Jour. de physiol. et de path. gén. 3, 703, 1901.
94. Doyon, M., et Kareff, M.: Action de l'adrénaline sur le glycogène du foie.—Compt. rend. de la soc. de biol. 56, 66, 1904.
95. Dupuytren et Thénard: Sur le diabète Sucré.—Bulletin de la société de la faculté de méd. 1, 37, 1806.
96. Dwight, E.: The Significance of Albumin and Casts, When Found in the Urine of Apparently Healthy Applicants for Life Insurance.—Proc. of the 16th Annual Meet. of Life Ins. Med. Dir. 1905, 393.
97. Eckhard, C.: Ueber den Morphindiabetes.—Beiträge zur Anat. und Physiologie. 8, 77, 1879.
98. Edie, E., and Spence, D.: Improved Method for the Determination of Sugar in Blood and Other Tissues with a Consideration of the Condition of the Sugar in the Blood.—Biochem. Jour. 2, 103, 1907.
99. Edwards, D., Page, I., and Brown, R.: Some Cardio-Vascular Changes Accompanying Insulin Hypoglycemia.—Proc. of the Soc. for Exp. Biol. and Med. 21, 170, 1924.
100. Ege, R.: Zur Frage der Permeabilität der Blutkörperchen gegenüber Glucose und Anelektrolyten.—Biochem. Zeitschr. 107, 246, 1920.
101. Embden, G., Lüthje, H., and Liefmann, E.: Über den Einfluss der Aussentemperatur auf den Blutzuckergehalt.—Beiträge zur chem. Phys. und Path. 10, 265, 1907.
102. Epstein, A.: An Accurate Microchemical Method of Estimating Sugar in the Blood.—Jour. of the Amer. Med. Assoc. 63, 1667, 1914.
103. Epstein, A., and Aschner, P.: The Effect of Surgical Procedures on the Blood Sugar.—Jour. of Biol. Chem. 25, 151, 1916.
104. Epstein, A., and Baehr, G.: Certain New Principles Concerning the Mechanism of Hyperglycaemia and Glycosuria.—Jour. of Biol. Chem. 18, 21, 1914.
105. Epstein, A., Reiss, J., and Branower, J.: The Effect of Surgical Procedures on Blood Sugar and Renal Permeability.—Jour. of Biol. Chem. 26, 25, 1916.
106. Erlandson, A.: Experimentelle Untersuchungen über den Phlorhizindiabetes.—Biochem. Zeitschr. 23, 329, 1909-10.
107. Exton in Discussion of Paper by Joslin: Diabetes and Life Insurance.—Transactions of the 32nd Annual Meeting of the Assoc. of Life Ins. Med. Directors, 1921, 107.
108. Falta, W., and Priestley, J.: Beiträge zur Regulation von Blutdruck und Kohlenhydrat-Stoffwechsel Durch das chromaffine System.—Berl. klin. Wochenschr. 48, 2102, 1911.
109. Falta, W., und Richter-Quittner, M.: Über die Verteilung des Zuckers, der Chloride und der Reststickstoffkörper auf Plasma und Körperchen im strömenden Blute.—Biochem. Zeitschr. 100, 148, 1919.

110. Fehling: Quantitative Bestimmung des Zucker im Harn.—Arch. für physiol. Heilkunde, 7, 64, 1848.
111. Fehling, H.: Die quantitative Bestimmung von Zucker und Starkmehl mittelst Kupfervitriol.—Ann. der Chem. und Pharm., 72, 106, 1849.
112. Finlay, F., and Rabinovitch, I.: Renal Glycosuria: A Clinical and Metabolic Study.—Quarterly Jour. of Med. 17, 26, 1924.
113. Fiquier, L.: Mémoire sur l'origine du sucre contenu dans le foie, et sur l'éxistence normale du sucre dans le sang de l'homme et des animaux dans le foie.—Gazette hédomadaire de méd et de chirurgie 2, 82 and 236, 1855.
114. Fiquier, L.: Troisième mémoire à propos de la fonction glycogénique du foie.—Gaz. hédomad de méd et de chirurg. 2, 634, 1855.
115. Fischer, E.: Verbindungen des Phenylhydrazin mit der Zuckarten.—Ber. der deutsch. chem. Gesellsch. 17, 579, 1884.
116. Fisher, E.: Über die Struktur der beiden Methylglukoside und über ein drittes Methylglukoside.—Ber. der deutsch. chem. Gesellsch. 47, 1900, 1914.
117. Fisher, G., and Wishart, M.: Animal Calorimetry. Fourth Paper. Observation on the Absorption of Dextrose and the Effect it has upon the Composition of the Blood.—Jour. of Biol. Chem. 13, 49, 1912.
118. Fisher, J.: Mortality on Accepted Risks Involving Albuminuria and Glycosuria.—Proc. of the 29th Ann. Meet. of the Assoc. of Life Ins. Med. Directors, 1918, 214.
119. Fisk, E.: Preventable Diseases of Adult Life.—New York State Jour. of Med. 1921, Mar. 23, p. 9.
120. Fitz, R.: A Comparison of Bang's Micro-Method for Determining Blood Sugar with Bertrand's Method.—Arch. of Int. Med. 14, 133, 1914.
121. Fitz, R., in Discussion of Sherrill's Paper.—Jour. of the Amer. Med. Assoc. 77, 1784, 1921.
122. Fitz, R., and Bock, A.: The Total Amount of Circulating Sugar in the Blood in Diabetes Mellitus and other Conditions.—Jour. of Biol. Chem. 48, 313, 1921.
123. Fletcher, A., and Campbell, W.: The Blood Sugar Following Insulin Administration and the Symptom Complex-Hypoglycemia.—Jour. of Metab. Rec. 2, 638, 1922.
124. Folin, O., and Berglund, H.: Some New Observations and Interpretations with Reference to Transportation, Retention and Excretion of Carbohydrate.—Jour. of Biol. Chem. 51, 213, 1922.
125. Folin, O., Denis, W., and Smilie, W.: Some Observations on "Emotional Glycosuria" in Man.—Jour. of Biol. Chem. 17, 519, 1914.
126. Folin, O., and Wu, H.: A System of Blood Analysis.—Jour. of Biol. Chem. 38, 81, 1919.
127. Folin, O., and Wu, H.: A System of Blood Analysis, 1. A Simplified and Improved Method for the Determination of Sugar.—Jour. of Biol. Chem. 41, 367, 1920.
128. Forrest, W., Smith, W., and Winter, L.: On the Change in the Nature of the Blood Sugar Caused by Insulin.—Jour. of Physiol. 57, 224, 1923.
129. Foster, G.: Studies on Carbohydrate Metabolism. II. An Interpretation of the Blood Sugar Phenomena Following the Ingestion of Glucose.—Jour. of Biol. Chem. 55, 303, 1923.

212 Thirty-Fifth Annual Meeting

130. Frank, C.: Über experimentelle und klinische Glykosurie renalen Ursprungs.—Arch. für exp. Path. und Pharmak. 72, 387, 1913.
131. Frank, E., and Isaac, S.: Beiträge zur Theorie experimenteller Diabetesformen.—Arch. für exp. Path. und Pharmak. 64, 309, 1910.
132. Frank, E., and Isaac, S.: Die Bedeutung des Adrenalin und des Cholin für die Erforschung des Zuckerstoffwechsel.—Zeitschr für exp. Path. und Ther. 7, 326, 1910.
133. Von Frerichs, F.: Ueber den Diabetes.—Berlin 1884.
134. Freund, H., and Marchand, F.: Über Blutzucker und Warmeregulation.—Arch. für exp. Path. und Pharmak. 73, 276, 1913.
135. Frey, E.: Menstruationstudien. I Zuckerstoffwechsel. Bemerkungen zu der gleichnamigen Arbeit von Heilig im Nr 14 dieser Wochenschr.—Klin. Wochenschr. 3, 1319, 1924.
136. Friedenwald, J., and Grove, G.: The Blood Sugar Tolerance Test as an Aid in the Diagnosis of Gastro-Intestinal Cancer.—Amer. Jour. of Med. Sci. 160, 313, 1920.
137. Friedenwald, J., and Grove, G.: Further Observations on the Blood Sugar Tolerance Test as an Aid in the Diagnosis of Gastro-Intestinal Cancer.—Amer. Jour. of Med. Sci. 163, 33, 1922.
138. Frost, H. M.: A Study of Cardio-vascular Reaction to Abnormal Variations of Intrathoracic Pressure.—Trans. of the 33rd Annual Meet. of the Assoc. of Life Ins. Med. Directors, 9, 207, 1922.
139. Frost, H. : Report of Progress of the Application of the Cardio-Respiratory Test.—Trans. of the 34th Annual Meet. of the Assoc. of Life Ins. Med. Directors. 10, 232, 1923.
140. Galambos, A.: Über den renalen Diabetes.—Deutsch. med. Wochenschr. 40, 1301, 1914.
141. Garrod, A.: Lettsonian Lectures on Glycosuria.—Lettsonian Lecture III. Lancet. 1912, I, 483, 557, 629.
142. Garrod, A.: Discussion Non-Diabetic Glycosuria.—Brit. Med. Jour. 1913, 2, 850.
143. Gautrelet, J., et Thomas, L.: Chez le chien décapauisé l'excitation du splanchnique ne produit pas de glycosurie.—Compt. rend. de la soc. de biol. 67, 233, 1909.
144. Geelvink: Ueber alimentare Glykosurie bei Nervenkrankheiten.—Neurol. Centralbl. 17, 333, 1898.
145. Gettler, A., and Baker, W.: Blood Sugar.—Jour. of Biol. Chem. 25, 217, 1916.
146. Gilbert, A., et Badouin, A.: Sur la glycémie expérimentale.—Compt. rend. de la soc. de biol. 65, 710, 1908.
147. Gilbert et Carnot: Sur les rapports qui existent entre les quantités de glucose absorbés et éliminées.—Compt. rend. de la soc. de biol. 50, 330, 1898.
148. Glaessner, K.: Ueber Abkuhlungs-Glykosurie.—Wien. klin. Wochenschr. 19, 920, 1906.
149. Goetsch, E., Creslig, H., and Jacobson, C.: Carbohydrate Tolerance and the Posterior Lobe of the Hypophysis Cerebri.—Bull. of the Johns Hopkins Hosp. 22, 165, 1911.
150. Goetzky, F.: Physiologische und pathologische "Glykamische Reaktionen" des Sauglings.—Zeitschr für Kinderheilk. 27, 195, 1920-21.
151. Goodhard, J.: Clinical Remarks on Transient Glycosuria of Neurotic Origin.—Brit. Med. Jour. 1889 II 1381.

152. Goto, K.: Alimentary Renal Glycosuria.—Arch. of Int. Med. 22, 96, 1918.
153. Goto, K., and Kuno, N: Studies on Renal Threshold for Glucose.—Arch. of Int. Med. 27, 224, 1921.
154. Gradwohl, R., and Blaivas, A.: The Distribution of the Blood Sugar as Regards Corpuscles, Plasma and Whole Blood in Health and Disease in Man.—Jour. of Lab. and Clin. Med. 2, 416, 1916-17.
155. Graham, G.: Variations in the Blood Sugar in Health.—J. Physiol. 50, 285, 1915-1916.
156. Graham, G.: Diabetes Innocens.—Quarterly Jour. of Medicine. 10, 245, 1917.
157. Gray, H.: Blood Sugar Standards. Part I—Normal and Diabetic Persons.—Arch. of Int. Med. 31, 241, 1923.
158. Gray, H.: Blood Sugar Standards. Part II—In Conditions Neither Normal nor Diabetic.—Arch. of Int. Med. 31, 259, 1923.
159. Greenwald, I.: The Formation of Glucose from Citric Acid in Diabetes Mellitus and Phlorhizin Glycosuria.—Jour. of Biol. Chem. 18, 115, 1914.
160. Griffith, J.: Infantilism, with Two Cases, the Brissaud and the Fröhlich Types Respectively.—Amer. Jour. of Dis. of Children. 16, 103, 1918.
161. Grober, J.: Ueber den Einfluss von Muskelarbeit auf alimentaren Glykosurie.—Deutsch. Arch. für klin. Med. 95, 137, 1908-09.
162. Grote, L.: Blutzucker und Diatherapie bei Morbus Addisoni.—Münch. med. Wochenschr. 63, 1614, 1916.
163. Gumpertz, K.: Ueber die Beziehung zwischen Diabetes Mellitus und Hysterie.—Deutsch. med. Wochenschr. 22, 789, 1896.
164. Gyelin, H.: The Carbohydrate Metabolism in Hyperthyroidism as Determined by Examination of the Blood and Urine.—Arch. of Int. Med. 16, 975, 1915.
165. Haedke, M.: Ueber metatraumatische alimentare Glykosurie.—Deutsch. med. Wochenschr. 26, 501, 1900.
166. Hagedorn, H.: Einige Bemerkungen über die Verteilung der Glukose zwischen Blutkörperchen und Plasma.—Biochem. Zeitschr. 107, 248, 1920.
167. Hammam, D., and Hirschmann, I.: Alimentary Hyperglycemia and Glycosuria as a Test of Sugar Tolerance.—Proc. of the Assoc. of Amer. Phys. 31, 355, 1916.
168. Hammam and Hirschman: Studies on Blood Sugar. I. Alimentary Hyperglycemia as a Test of Sugar Tolerance.—Arch. of Int. Med. 20, 761, 1917.
169. Hammett, F.: The Practical Appreciation of Emotional Glycosuria.—Jour. of the Amer. Med. Assoc. 66, 1463, 1916.
170. Henriot, M.: Sur le sucre du sang.—Compt. rend. de la soc. de piol. Paris 50, 543, 1898.
171. Harle, F.: Hypertonie und Blutzucker.—Zeitschr für Klin Med. 92, 124, 1921.
172. Harley, V.: Ueber den physiologischen Abbau des Traubenzuckers.—Arch. für Physiol. 1893, Suppl. 46.
173. Harrison, G.: Glycosuria of Malarial Origin.—Brit. Med. Jour. 1921, 2, 630.
174. Haskins, H., and Holbrook, W.: Adaptation of Shaffer's Titration Method for Blood Sugar to Clinical Use.—Jour. of Lab. and Clin. Med. 8, 747, 1922-23.

175. Hasse: Vergiftung durch Kohlenoxydgas.—Schmidt's Jahrbuch der Medizin. 105, 41, 1860.
176. Hawk, P.: On the Influence of Ether Anesthesia.—Proc. of the Am. Physiol. Soc. Am. Jour. of Physiol. 10, p. xxxvii. 1903-4.
177. Hédon, M.: Sur la nature des sucres du sang.—Compt. rend. de la soc. de biol. Paris 50, 510, 1898.
178. Heilig, R.: Menstruationsstudien. I. Zuckerstoffwechsel.—Klin. Wochenschr. 3, 576, 1924.
179. Henriques, V.: Ueber reduzierende Stoffe des Blutes.—Zeitschr. für physiol. Chem. 23, 244, 1897.
180. Henriques, V., und Ege, R.: Vergleichende Untersuchungen über die Glukoskonzentration in dem arterielen Blut und in dem venösen Blut aus den Muskeln.—Biochem. Zeitschr. 119, 121, 1921.
181. Herrick, W.: Hypertension and Hyperglycemia.—Jour. of the Amer. Med. Assoc. 81, 1942, 1923.
182. Herter, C., and Richards, A.: Note on the Glycosuria Following Experimental Ingestion of Ardenalin.—Med. News. 80, 201, 1902.
183. Hirsh, E., und Reinbach, H.: Die Fesselungshyperglykamie und Fesselungsglykosurie des Kaninchens.—Zeitschr. für physiol. Chem. 87, 122, 1913.
184. Hirsch, E., und Reinbach, H.: Ueber "psychische" Hyperglykamie und Narkosehyperglykamie beim Hund.—Zeitschr. für physiol. Chem. 91, 292, 1914.
185. Höst, H., and Hatlehol, R.: Blood Sugar Concentration and Blood Sugar Method.—Jour. of Biol. Chem. 42, 347, 1920.
186. Hofmeister, F.: Ueber Resorption und Assimilation der Nahrstoff. Fünfte Mitteilung. Ueber die Assimilationagrenze der Zuckerkarten.—Arch. für exp. Path. und Pharmak. 25, 240, 1888-89.
187. Hohlweg, H.: Ueber den Einfluss der Muskelarbeit auf die Zersetzung subkutaner einverleibten Zuckers.—Zeitschr. für Biol. 55, 396, 1910-11.
188. Hohlweg, H., und Voit, F.: Ueber den Einfluss der Überheizung auf die Zersetzung des Zucker im Tierkörper.—Zeitschr. für Biol. 51, 491, 1908.
189. Hollinger, A.: Über Hyperglykämie bei Fieber.—Deutsch. Arch. für klin. Med. 92, 217, 1907-08.
190. Holst, J.: Studien über die alimentare Glykosurie.—Zeitschr. für klin. Med. 95, 394, 1922.
191. Hopkins, A.: Studies in the Concentration of Blood Sugar in Health and Disease as Determined by Bang's Micro-Method.—Am. J. M. Sc. 149, 254, 1915.
192. Hoppe-Seyler G.: Beiträge zur Kenntniss der Indigo-bildenden Substanzen im Harn und des künstlichen Diabetes Mellitus.—Zeitschr. für physiol. Chem. 7, 403, 1883.
193. Jacobi, C.: Ueber künstlichen Nierendiabetes.—Arch. für path. Jour. of Biol. Chem. 8, 112, 1922-23.
194. Irwin, J., Fyfe, A., and Hogg, T.: Derivatives of a New Form of Glucose.—Jour. of the Chem. Soc. 107, 524, 1915.
195. Jacobi, C.: Ueber Künstlichen Nierendiabetes.—Arch. für Path. Anat. und Pharmak. 35, 213, 1894-5.
196. Jacobsen, A.: Untersuchungen über den Einfluss des Chloralhydrate auf experimentelle Hyperglykämieformen.—Biochem. Ztschr. 51, 443, 1913.

197. Jacobsen, A.: Untersuchungen über den Einfluss verschiedener Nahrungsmittel auf den Blutzucker bei normalen zuckerkranken und graviden Personen.—Biochem. Ztschr. 56, 471, 1913.
198. Jacobsen, A., and Edwards, H.: Curves of Sugar and Urea after Standardized Protein Meals.—Amer. Jour. of Med. Sci. 159, 833, 1920.
199. Von Jaksch: Klinische Beiträge zur Kenntnis der alimentaren Glykosurie bei funktionelle Neurosen, Phosphorvergiftung und Leberatrophie.—Prag. med. Wochenschr. 20, 281, 1895.
200. Von Jaksch: Ueber die diagnostische Bedeutung der alimentaren Glykosurie.—Verhandl. des XIII Kongr. für inn. Med. 535, 1895.
201. Janney, N.: The Metabolic Relationship of Protein to Glucose.—Jour. of Biol. Chem. 20, 321, 1921.
202. Janney, N., and Isaacson, V.: A Blood Sugar Tolerance Test.—Proc. of the Soc. for Exp. Biol. and Med. 15, 15, 1917.
203. Janney, N., and Isaacson, V.: A Blood Sugar Tolerance Test.—Jour. of Amer. Med. Assoc. 70, 1131, 1918.
204. Janney, N., and Isaacson, J.: The Blood Sugar in Thyroid and Other Endocrine Diseases, The Significance of Hypoglycemia and the Delayed Blood Sugar Curve.—Arch. of Int. Med. 22, 160, 1918.
205. John, H.: The Interpretation of Blood Sugar Estimations That Are Near the Normal.—The Jour. of Lab. and Clin. Med. 8, 145, 1922.
206. John, H.: Differential Diagnosis of Diabetes by Means of Glucose Tolerance.—Jour. of Amer. Med. Assoc. 79, 1234, 1922.
207. John, H.: Glucose Tolerance and Its Value in Diagnosis.—Jour. of Metab. Res. 1, 497, 1922.
208. John, H.: Diabetes and Life Insurance.—Atlantic Medical Journal 26, 539, 1923.
209. John, H.: Differential Diagnosis of Diabetes.—Amer. Jour. of Med. Sciences 166, 275, 1923.
210. John, H.: Distribution of Sugar in Whole Blood, Plasma and Corpuscles; Permeability of Red Blood Corpuscles for Sugar in Diabetic and Nondiabetic Cases.—Arch. of Int. Med. 31, 555, 1923.
211. John, H.: Insulin in the Treatment of Diabetes.—Medical Jour. and Rec. 119, 229, 1924.
212. John, H.: One Relation of Glycosuria to Kidney Permeability.—Endocrinology 7, 699, 1923.
213. John, H.: The Relationship of Blood Sugar Content to Kidney Permeability and Glycosuria.—Jour. of Lab. and Clin. Med. 9, 626, 1924.
214. Jones, M.: Effect of Carbohydrate Feeding on Blood Sugar.—Jour. of Biol. Chem. 43, 507, 1920.
215. Joslin, E.: The Causes of Death in Diabetes.—Amer. Jour. of Med. Sci. 151, 313, 1916.
216. Joslin, E.: Diabetes and Life Insurance.—Transactions of the 32nd Annual Meeting of the Assoc. of Life Ins. Med. Directors, 1921, 87.
217. Joslin, E.: The Prevention of Diabetes Mellitus.—Jour. of the Amer. Med. Assoc. 76, 79, 1921.
218. Joslin, E.: The Treatment of Diabetes.—3rd Ed. N. Y., 1923.

216 Thirty-Fifth Annual Meeting

219. Junkersdorf, P.: Über den Einfluss der Phlorhizinvergiftung auf den Zuckergehalt des Blutes.—Arch. für die gesamt. Physiol. 131, 306, 1910.
220. Kahn, M.: A Clinical Method for the Quantitative Estimation of Small Amounts of Blood.—Jour. of the Amer. Med. Assoc. 64, 241, 1915.
221. Kahn, R.: Zur Frage nach der inneren Sekretion des chromaffinen Gewebes.—Arch. für die gesamt. Physiol. 128, 519, 1909.
222. Kahn, S.: Reduction of Human Blood Sugar by Means of Insulin.—Boston Med. and Surg. Jour. 191, 161, 1924.
223. Kahn and Starkenstein: Über das Verhalten des Glykogens nach Nebennierenextirpation.—Arch. für die gesamt. Physiol. 139, 181, 1911.
224. Kern, R., and Jonas, L.: Sugar Tolerance Testing. Importance of Blood Sugar Curve During the First Hour After Glucose Meal.—Jour. of the Amer. Med. Assoc. 81, 1439, 1923.
225. Kleiner, I.: The Disappearance of Dextrose from the Blood After Intravenous Injection.—Jour. of Exp. Med. 23, 507, 1910.
226. Kleiner, I.: A Clinical Method for the Determination of Blood Sugar in Minute Quantities of Blood.—Jour. of the Amer. Med. Assoc. 76, 172, 1921.
227. Klemperer: Ueber regulatorische Glykosurie und renalen Diabetes, 1896.
228. Klemperer: Verhandl. der Ver. für inn. Med. zu Berl. 16, 67, 1896.
229. Klemperer: Sitzung des Verein für innere Medizin zu Berlin 18 Mai. und 19 Juni. 1896.
230. Knight: President's Address.—Transactions of the 32nd Annual Meeting of the Assoc. of Life Ins. Med. Directors, 1921, 6.
231. Kramer, B., and Coffin, H.: The Role of Psychic and Sensory Stimuli in the Hyperglycemia Produced by Lowering the Environmental Temperature of Dogs.—Jour. of Biol. Chem. 25, 423, 1916.
232. Kramer, B., and Coffin, H.: Stimuli and Hyperglycemia.—Jour. of Biol. Chem. 25, 426, 1916.
233. Kramer, B., and Gittleman, J.: Technique for Quantitative Estimation of Sugar in Very Small Amounts of Blood.—Jour. of Amer. Med. Assoc. 81, 1171, 1923.
234. Kraus, F., and Ludwig, H.: Klinische Beiträge zur alimentare Glycosurie.—Wein. klin. Wochenschr. 4, 855 and 897, 1891.
235. Kulz, C.: Beiträge zur Hydrurie und Melliturie.—Eckhardt's Beiträge zur Anat. und Physiol. 6, 117, 1872.
236. Kuriyama, S.: The Influence of Intravenous Injection of Witte's Peptone upon the Sugar Content of the Blood and Epinephrine Hyperglycemia and Glycosuria.—Jour. of Biol. Chem. 29, 127, 1917.
237. Kuriyama, S.: The Adrenals in Relation to Carbohydrate Metabolism. I. The Influence of Repetition of Epinephrine Injection upon the Intensity of Glycosuria and Hyperglycemia and the Glycogen Content of the Liver.—Jour. of Biol. Chem. 34, 269, 1918.
238. Kylin, E.: Studien über das Hypertonie-Hyperglykämie-Hyperurikamiesyndrome.—Zeitschr. für inn. Med. 44, 81, 1923.
239. Labbé, M. et Debré, R.: Diabète post-ourlien transitoire.—Bull. med. 35, 600, 1921.

241. Laudenheimer, R.: Diabetes und Geisterstörung.—Berl. klin. of Med. Sci. 157, 201, 1919.
241. Laudenheimer, R.: Diabetes und Geisterstörung.—Berl. Klin. Wochenschr. 35, 463, 1898.
242. Lavesson, H.: Beitrag zur Bestimmung der reduzierenden Stoffe im Normalen Harn.—Biochem. Zeitschr. 4, 40, 1907.
243. Lazarus, P.: Experimentelle Hypertrophie der Langerhans'schen Pankreasinseln bei der Phlorizinglykosurie.—Munch. med. Wochenschr. 34, 2222, 1907.
244. Von Leersum, E., und Polenai, J.: Ist Phlorhizin instande, Hypertrophie und Hyperplasie der Langerhans'schen Pankreasinseln Hervorzuufen.—Arch. für exp. Path und Pharmacol. 62, 266, 1910.
245. Lemaire, F.: Ueber das Vorkommen von Milchzucker im Harn bei Wöchnerinnen.—Zeitschr. für physiol. Chem. 21, 442, 1895-6.
246. Lépine, R.: La glycosurie phlorhizique. Revue critique.—Arch. de méd. expérimentale, 13, 710, 1901.
247. Lépine, R.: Le diabète sucré.—Paris, 1909, 193.
248. Lépine, R., et Bouland: Sur l'absence d'hyperglycémie dans la glycosurie uranique.—Compt. rend. de la soc. de biol. 55, 1289, 1903.
249. Lépine, R., et Bouland: Sur l'absence d'hyperglycémie dans la glycosurie uranique.—Rev. de méd. 1904, 24.
250. Leudet, E.: Recherches cliniques sur l'influence des maladies cérébrales.—Compt. rend. de la soc. de biol. sér. 2, Vol. 4, 123, 1857.
251. Levene, P.: Studies in Phlorhizin Glycosuria.—Jour. of Physiol. 17, 259, 1894-95.
252. Levinstein, F.: Die Morphiumsucht.—Berlin klin. Wochenschr. 12, 447, 1875.
253. Levinstein: Zur Pathologie der acuten Morphium—und acuten Chloral—Vergiftungen.—Berl. klin. Wochenschr. 13, 388, 1876.
254. Lewandowsky, F.: Zur Kenntnis des Phlorhizindiabetes.—Arch. für Physiol. 1901, 365.
255. Lewis, D.: Renal Glycosuria.—Arch. of Int. Med. 29, 418, 1922.
256. Lewis, R., and Benedict, S.: A Method for the Estimation of Sugar in Small Quantities of Blood.—Jour. of Biol. Chem. 20, 61, 1915.
257. Lewis, D., and Mosenthal, H.: Renal Diabetes.—Bull. of the Johns Hopkins Hospital 27, 133, 1916.
258. Liefmann, E., and Stern, B.: Über Glykaemie und Glykosurie.—Biochem. Zeitschr. 1, 299, 1906.
259. Linossier, S., et Roque, G.: Contribution a l'étude de la glycosurie alimentaire.—Arch. de méd. expérimentale. 7, 228, 1895.
260. Lüthje, H.: Beitrag zur Frage des renalen Diabetes.—Münch. med. Wochenschr. 48, 1471, 1901.
261. Lüthje: Ueber den Einfluss der Aussentemperatur auf die Grösse der Zuckerausscheidung.—Verhandl. des 22 Kongr. für inn. Med. 1905, 268.
262. Lüthje: Beitrag zur Frage der Zuckerökonomie im Tierkörper.—Verhandl. der 24 Kongr. für inn. Med. 1907, 264.
263. Lusk, G.: The Influence of Cold and Mechanical Exercise on the Sugar Excretion in Phlorhizin Glycosuria.—Amer. Jour. of Physiol. 22, 163, 1908.

218 Thirty-Fifth Annual Meeting

264. Lyman, R., Nichols, E., and McCann, W.: The Respiratory Exchange and Blood Sugar Curves of Normal and Diabetic Subjects After Epinephrin and Insulin.—*Jour. of Pharm. and Exp. Ther.* 21, 343, 1923.
265. MacLean, H.: On the Influence of Creatinin in Modifying Certain Reactions of Sugar in Urine.—*Biochem. Jour.* 2, 156, 1907.
266. MacLean, H.: Glycolysis in Diabetic Blood with a Method for the Estimation of Blood Sugar.—*Jour. of Physiol.* 50, 168, 1915-16.
267. MacLean, H.: On the Estimation of Sugar in Blood.—*Biochem. Jour.* 13, 135, 1919.
268. MacLean and de Wesselow: The Estimation of Sugar Tolerance.—*Quarterly Jour. of Med.* 14, 103, 1920-21.
269. MacLeod, J.: Diabetes. Its Pathological Physiology.—N. Y. Longmans, Green & Co. 1913.
270. MacLeod, J.: Recent Work on the Physiologic Pathology of Glycosuria.—*Jour. of the Amer. Med. Assoc.* 62, 1222, 1914.
271. MacLeod, J.: A Rapid and Accurate Clinical Method for the Estimation of Sugar in Small Quantities of Blood.—*Jour. of Lab. and Clin. Med.* 1, 445, 1915-16.
272. MacLeod, J., Christie, C., and Donaldson, J.: The Estimation of Dextrose in Blood and Urine by the Difference in Reducing Power Before and After Yeast Fermentation.—*Proceed. of the Amer. Soc. of Biol. Chem.*, 1911, p. xxvi, vol. 11, *Jour. of Biol. Chem.*
273. MacLeod, J., and Pearce: Studies in Experimental Glycosuria. VIII. The Relationship of the Adrenal Gland to Sugar Production by the Liver.—*Amer. Jour. of Physiol.* 29, 419, 1912.
274. MacLeod, J., and Wedd, A.: The Behavior of the Sugar and Lactic Acid in the Blood Flowing from the Liver, After Temporary Occlusion of the Hepatic Pedicle.—*Jour. of Biol. Chem.* 18, 447, 1914.
275. MacNider, W.: A Study of the Action of Various Diuretics in Uranium Nephritis.—*Jour. of Pharmac. and Exp. Therap.* 3, 423, 1911-12.
276. MacNider, W.: A Study of the Action of Various Diuretics in Uranium Nephritis, with Especial Reference to the Part Played by the Anesthetics in Determining the Efficiency of the Diuretic.—*Jour. of Pharmac. and Exp. Therap.* 4, 491, 1912-13.
277. Marsh, P.: Renal Glycosuria.—*Arch. of Int. Med.* 28, 54, 1921.
278. Marsh, P.: The Significance of Glycosuria.—*Jour. of Lab. and Clin. Med.* 9, 663, 1923.
279. Mayer, A.: Sur le mode d'action de la piqûre diabétique, rôle des capsules surrénales.—*Compt. rend. de la soc. de biol.* 60, 1123, 1908.
280. McCrudden, F.: Metabolism in Diseases of the Neuromuscular System. Endocrinology and Metabolism.—Edited by Barker, N. Y. 1922, vol. IV, p. 707.
281. McDanell, L., and Underhill, F.: The Influence of Alkali Administration upon Blood Sugar Content in Relation to the Acid-base-producing Properties of the diet.—*Jour. of Biol. Chem.* 29, 227, 1917.
282. McDanell, L., and Underhill, F.: The Influence of Acid-forming and Base-forming Diets upon Blood Sugar Content.—*Jour. of Biol. Chem.* 29, 233, 1917.

283. McDanell, L., and Underhill, F.: The Relation of Epinephrine Glycosuria to Dosage and to the Character of the Diet.—*Jour. of Biol. Chem.* 29, 245, 1917.
284. McDanell, L., and Underhill, F.: Further Experiments upon the Influence of the Intravenous Injection of Sodium Carbonate upon Epinephrine Hyperglycemia and Glycosuria.—*Jour. of Biol. Chem.* 29, 251, 1917.
285. McDanell, L., and Underhill, F.: New Experiments upon the Mechanism of Salt Glycosuria.—*Jour. of Biol. Chem.* 29, 273, 1917.
286. McGregor, R.: An Experimental Inquiry into the Comparative State of Urine in Healthy and Diseased Urine and the Seat of the Formation of Sugar in Diabetes Mellitus.—*London Medical Gazette* 2, 268, 1837.
287. McGuigan, H., and Ross, E.: Methods for the Determination of Blood Sugar in Reference to Its Condition in the Blood.—*Jour. of Biol. Chem.* 31, 533, 1917.
288. McLean, F.: The Sugar Content of the Blood and Its Clinical Significance.—*Jour. of the Amer. Med. Assoc.* 62, 917, 1914.
289. Medico-Actuarial Investigation.—4 (Part I), 45, 1914.
290. Von Mehring: Über experimentellen Diabetes.—*Berhandl der VI Kongr für inn. Med.* Wiesbaden, 1886, 185.
291. Von Mehring, J.: Über Diabetes Mellitus II.—*Zeitschr. für klin. Med.* 16, 431, 1889.
292. Mendel, L., and Fine, M.: Studies in Nutrition. I. The Utilization of the Proteins of Wheat.—*Jour. of Biol. Chem.* 10, 303, 1911-12.
293. Mendel, L., and Fine, M.: Studies in Nutrition. II. The Utilization of the Protein of Barley.—*Jour. of Biol. Chem.* 10, 339, 1911-12.
294. Mendel, L., and Fine, M.: Studies in Nutrition. III. The Utilization of the Protein of Corn.—*Jour. of Biol. Chem.* 10, 345, 1911-12.
295. Mendel, L., and Jones, M.: Studies on Carbohydrate Metabolism in Rabbits. I. Observations on the Limits of Assimilability of Various Carbohydrates.—*Jour. of Biol. Chem.* 43, 491, 1920.
296. Mendel, L., and Lewis, R.: The Rate of Elimination of Nitrogen as Influenced by Diet Factors. I. The Influence of the Texture of the Diet.—*Jour. of Biol. Chem.* 16, 19, 1913-14.
297. Mendel, L., and Lewis, R.: The Rate of Elimination as Influenced by Diet Factors. II. The Influence of Carbohydrate and Fats on the Diet.—*Jour. of Biol. Chem.* 16, 37, 1913-14.
298. Mendel, L., and Lewis, R.: The Rate of Elimination of Nitrogen as Influenced by Diet Factors.—*Jour. of Biol. Chem.* 16, 55, 1913-14.
299. Mertz, A., und Rominger, E.: Experimentelle Blutzuckeruntersuchungen bei Kindern.—*Arch. für Kinderheilkunde* 69, 81, 1921.
300. Mesing, E.: Sind die roten Blutkörper durchgangig für Traubenzucker?—*Arch. für die gesamt. Physiol.* 149, 227, 1912-13.
301. Metzger, L.: Zur Lehre von Nebennierendiabetes.—*Münch med. Wochenschr.* 49, 478, 1902.
302. Michaud: Ueber die Kohlenhydratstoffwechsel bei Hunden mit Eck'schen Fistel.—*Verhandl. des Kongr. für inn. Med.* 28, 561, 1911.

303. Minkowski, O.: Untersuchungen über den Diabetes Mellitus nach Extirpation des Pankreas.—Arch. für exp. Path. und Pharmak. 31, 85, 1892-3.
304. Mita, S.: Beitrag zur Kenntnis der Glykosurie bei Geisteskranken.—Monatsheft. für Psych. und Neurol. 32, 159, 1912.
305. Moore, J.: Liquor Potassae Test for Sugar in the Urine.—Lancet, 1844, 1, 75.
306. Morat, J., et Dufourt: Consommation du sucre par les muscles.—Arch. de Physiol. 24, 327, 1892.
307. Morgulis, S., Edwards, A., and Leggett, E.: A Modification of the Folin-Wu Blood Sugar Method.—Jour. of Lab. and Clin. Med. 8, 339, 1922-23.
308. Morgulis, S., and Jahr, H.: Note on the Lewis-Benedict Method of Blood Sugar Determination.—Jour. of Biol. Chem. 39, 119, 1919.
309. Moritz, F.: Ueber die Kupferoxyd-reduzierenden Substanzen des Harnes unter physiologischen und pathologischen Verhältnissen.—Deutsch. Arch. für klin. Med. 46, 217, 1889-90.
310. Moritz, F.: Ueber alimentare Glycosurie.—Kongress. für inn. Med. 10, 492, 1891.
311. Morris, M.: Value of the Alimentary Test in the Diagnosis of Mild Hyperthyroidism.—Jour. of the Amer. Med. Assoc. 76, 1566, 1921.
312. Morrison, N., and Ohler, W.: Blood Sugar and Race.—Boston Med. and Surg. Jour. 188, 852, 1923.
313. Mozotowski, W.: Sur la nature du sucre sanguin.—Compt. rend. de la soc. de biol. 90, 311, 1924.
314. Müller, W.: Der Nachweis des Zuckers im Harne mittelst Kupferoxyde und alkalische Seignettesalzlösung.—Arch. für die gesamte Physiol. 27, 107, 1882.
315. Müller, W.: Die Ausscheidung des Zuckers im Harne des gesunden Menschen nach Genuss von Kohlenhydraten.—Arch. für die gesamte Physiol. 34, 576, 1884.
316. Muhlberg, W.: A Few Practical Observations. The Result of 8,000 Examinations of Urine.—Transactions of the 24th Annual Meeting of the Assoc. of Life Ins. Medical Directors, 1913, 183.
317. Muhlberg: Discussion of Joslin's Paper. Diabetes and Life Insurance.—Transactions of the 32nd Annual Meeting of the Assoc. of Life Insurance Medical Directors, 1921, 100.
318. Murlin, J., and Kramer, B.: The Influence of Pancreatic and Duodenal Extracts on the Glycosuria and the Respiratory Metabolism of Depancreatized Dogs.—Jour. of Biol. Chem. 15, 365, 1913.
319. Murlin, J., and Kramer, B.: The Influence of Alkali and Acid upon the Glycosuria and Hyperglycemia.—Jour. of Biol. Chem. 27, 481, 1916.
320. Myers, V.: A Method for the Determination of Small Amounts of Sugar in Urine.—Proc. of the Soc. for Exp. Biol. and Med. 13, 178, 1916.
321. Myers, V.: Chemical Changes in the Blood in Disease. IV. Blood Sugar.—Jour. of Lab. and Clin. Med. 5, 640, 1920.
322. Myers, V., and Bailey, C.: The Lewis and Benedict Method for the Estimation of Blood Sugar, with Some Observations Obtained in Disease.—Jour. of Biol. Chem. 24, 147, 1916.
323. Nakayama, M.: Über den normalen Wert der Zuckerausscheidungsschwelle.—Jour. of Biochem. (Japan) 3, 407, 1924.

324. Naunyn, B.: Der Diabetes Mellitus.—Wien 1906. 2te Aufl.
325. Neubauer, E.: Über Hyperglykämie bei Hochdrucknephritis und die Beziehung zwischen Glykämie und Glucosurie beim Diabetes Mellitus.—Biochem. Zeitschr. 25, 284, 1910.
326. Neuworth, I., and Kliener, I.—The Blood Sugar Content of Capillary Blood as Compared with that of Venous Blood.—Jour. of Lab. and Clin. Med. 7, 495, 1921-22.
327. Nishi, M.: Über den Mechanismus der Diuretinglykosurie.—Arch. für exp. Path. und Pharmak. 61, 401, 1909.
328. Nonnenbruch, W., and Szyszka, W.: Über die Veränderungen im Blut und Harn nach intravenöser Zuckeraufinfusionen beim Menschen.—Arch. für exp. Path. und Pharmak. 86, 281, 1920.
329. Von Noorden, C.: Die Zuckerkrankheit und ihre Behandlung.—Berlin, 1917, 7th Ed.
330. Novak, J., Porges, O., und Strisower, R.: Ueber Nierendiabetes in der Gravidität.—Deutsch. med. Wochenschr. 38 (2), 1868, 1912.
331. Nylander, E.: Ueber alkalische Wismutlösungen als Reagenz auf Traubenzucker im Harne.—Zeitschr. für physiol. Chem. 8, 175, 1884.
332. Ogden, J.: A Study of 54,270 Exposures of Ordinary Life Insurance Showing Urinary Impairments at the time of Acceptance.—Transactions of the 26th Annual Meeting of the Assoc. of Life Ins. Medical Directors, 1915, 132.
333. Ohler, W.: Blood Sugar at Annual Intervals.—Med. Clin. of North America 5, 1465, 1921-22.
334. Olmstead, W., and Gay, L.: Study of Blood Sugar Curves Following a Standardized Glucose Meal.—Arch. of Int. Med. 29, 384, 1922.
335. Oppler, B.: Die Bestimmung des Traubenzucker im Harn und Blut.—Zeitschr. für physiol. Chem. 75, 71, 1911.
336. Oppler, B., und Rona, F.: Untersuchungen über Blutzucker III.—Biochem. Zeitschr. 13, 121, 1908.
337. Van Ordt: Alimentare Glykosurie bei Krankheiten des Zentralnervensystems.—Münch. med. Wochenschr. 45, 2, 1898.
338. Page, I.—On the Normal Urine Sugar Curve in Normal Individuals, Borderline Diabetics and Severe Diabetics under Insulin Treatment.—Jour. of Lab. Med. 8, 631, 1923.
339. Palmer, W.: The Concentration of Dextrose in the Tissues of Normal and Diabetic Animals.—Jour. of Biol. Chem. 30, 79, 1917.
340. Patton, J.: The Importance of Glycosuria in Life Insurance.—Transactions of the 27th Annual Meeting of the Assoc. of Life Ins. Medical Directors, 1916, 406.
341. Pauli in Discussion of Paper by Blakely: The Importance of Glycosuria in Life Insurance.—Transactions of the 27th Annual Meeting of the Assoc. of Life Ins. Medical Directors, 1916, 479.
342. Pauli in Discussion of Paper by Wolf: A Study of the More Common Methods Used to Detect Sugar in Urine with Special Reference to the Tests Employed in the Laboratory of the Equitable Life Assurance Society.—Transactions of the 31st Annual Meeting of the Assoc. of Life Insurance Medical Directors, 1920, 327.
343. Paullin, J.: Renal Glycosuria.—Jour. of the Amer. Med. Assoc. 75, 214, 1920.

Thirty-Fifth Annual Meeting

344. Paullin, J.: Glucose Tolerance Test in the Obese.—*Jour. of the Amer. Med. Assoc.* 77, 1996, 1921.

345. Pavy, F.: On the Recognition of Sugar in Healthy Urine.—*Guy's Hospital Reports, 3rd Series*, 21, 413, 1876.

346. Pavy, F.: On Phloridzin Diabetes.—*Jour. of Physiol.* 20, Proc. p. xix, 1896.

347. Pavy, F.: An Inquiry into the Effects on the Blood and Urine of the Intravenous and Subcutaneous Ingestion of Various Carbohydrates Standing in Relation to Animal Life.—*Jour. of Physiol.* 24, 479, 1899.

348. Pavy, F.: On the Nature of the Sugar Present in Normal Blood, Urine and Muscle.—*Jour. of Physiol.* 26, 282, 1900.

349. Pavy, F., Brodie, T., and Siau, R.: On the Mechanism of Phloridzin Glycosuria.—*Jour. of Physiol.* 29, 467, 1903.

350. Pearce, R.: A Criticism of the Bang and Lewis-Benedict Methods for the Estimation of Blood Sugar with Suggestions for a Modification of the Latter Method.—*Jour. of Biol. Chem.* 22, 525, 1915.

351. Pels, I.: The Sugar Content of the Blood in Certain Diseases of the Skin.—*Jour. of the Amer. Med. Assoc.* 65, 2077, 1915.

352. Pemberton, R., and Foster, G.: Studies on Arthritis in the Army Based on Four Hundred Cases. III. Studies on the Nitrogen, Total Fat, and Cholesterol of the Fasting Blood, Renal Function, Blood Sugar, and Sugar Tolerance.—*Arch. of Int. Med.* 25, 243, 1920.

353. Philipeaux, J., et Vulpian, A.: Note sur un cas de diabète passager survenu pendant le cours du développement d'un anthrax.—*Gazette hédomadaire de méd. et de chirurgie*. 8, 782, 861.

354. Pickhardt, M.: Der Nachweis von Traubenzucker im Blut.—*Zeitschr. für physiol. Chem.* 71, 217, 1893.

355. Porcher, C.: Des injections de phlorizine chez la vache laitière.—*Compt. rend. de la soc. de biol.* 138, 1457, 1904.

356. Purjesz, B.: Der Blutzuckergehalt unter normalen und pathologischen Verhältnisse.—*Wien. klin. Wochenschr.* 26, 1420, 1913.

357. Quinquaud, C.: Expérience sur la contraction musculaire et la chaleur animale.—*Compt. rend. de la soc. de biol.* 38, 410, 1886.

358. Quinquaud, C.: De la glycosurie physiologique.—*Compt. rend. de la soc. de biol.* 1889, 349.

359. Rabinovitz, I.: Observations on the Effects of Insulin in the Treatment of Diabetes Mellitus.—*Arch. of Int. Med.* 32, 796, 1923.

360. Raimann: Ueber alimentare Glykosurie. Zweite Mitteilung. Glykosurie und alimentare Glykosurie bei Geisteskranken.—*Wien. klin. Wochenschr.* 14, 512, 1901.

361. Raimann, E.: Ueber Glykosurie und alimentare Glykosurie bei Geisteskranken.—*Zeitschr. für Heilkunde*. 23, Abth. 3, p. 1 and 145, 1902.

362. Rakestraw, N.: Chemical Factors in Fatigue. I. The Effect of Muscular Exercise Upon Certain Common Blood Constituents.—*Jour. of Biol. Chem.* 47, 565, 1921.

363. Rakestraw, N.: Chemical Factors in Fatigue. II. Further Changes in Some of the Constituents Following Strenuous Exercise.—*Jour. of Biol. Chem.* 56, 121, 1923.

364. Raphael, F.: Untersuchungen über alimentare Glykosurie.—*Zeitschr. für klin. Med.* 37, 19, 1899.
365. Reicher, K., and Stein, E.: Beitrage zur Physiologie und Pathologie des Kohlenhydratstoffwechsel.—*Biochem. Zeitschr.* 37, 321, 1911.
366. Richardson: Lecture on Diabetes.—*Medical Times and Gazette* 1, 233, 1862.
367. Richter: Diuretin und Glycosurie—*Zeitschr. für klin. Med.* 35, 463, 1898.
368. Richter, P.: Zur Frage des "Nierendiabetes."—*Deutsche med. Wochenschr.* 1899, II 840.
369. Rigler, L. G., and Ulrich, H. L.: Blood Sugar Reaction Following Intravenous Injection of Glucose.—*Arch. of Int. Med.* 32, 343, 1923.
370. Ringer, A.: The Influence of Ardenalin in Phlorhizin Diabetes.—*Proc. of the Soc. for Exp. Biol. and Med.* 7, 8, 1909-10.
371. Ritter, F.: Ueber das Amylum und den Zucker des Leber.—*Zeitschr. für rationelle Med.* III Reihe 24, 65, 1865.
372. Röhmann, F.: Ueber die Bestimmung des Zuckers im Blut.—*Centralbl. für Physiol.* 4, 12, 1890-91.
373. Rogers, O.: The Effect of Glycosuria and of Albuminuria on Mortality.—*Transactions of the 28th Annual Meeting of the Assoc. of Life Insurance Medical Directors*, 1917, 34.
374. Rona, P., und Michaelis, L.: Untersuchungen über den Blutzucker.—*Biochem. Zeitschr.* 7, 329, 1907.
375. Rona, P., and Michaelis, L.: Über die Absorption des Zuckers.—*Biochem. Zeitschr.* 16, 489, 1909.
376. Rose, U.: Der Blutzuckergehalt der Kaninchen, seine Erhöhung durch den Aderlass, durch die Eröffnung der Bauchhöhle und durch die Nierenausschaltung und seine Verhalten im Diuretindiabetes.—*Arch. für exp. Path. und Pharmak.* 50, 15, 1903.
377. Rosin, H.: Ueber die quantitativen Verhältnisse der Kohlenhydrate im diabetischen Harn.—*Deutsch. med. Wochenschr.* 1900, II 497.
378. Ross, E.: Blood Dextrose as Affected by Morphine and Morphine with Ether Anesthesia.—34, 335, 1918.
379. Ross, E., and Hawk, P.: Post-anesthetic Glycosuria as Influenced by Diet, Body Temperature and Purity of the Ether.—*Arch. of Int. Med.* 14, 779, 1914.
380. Sakaguchi, K.: Beitrage zur Diabetesforschung. Erste Mitteilung. Über den Einfluss von Menge, Art und Zeit der Kohlenhydratzufuhr auf den Blutzuckergehalt beim gesunden Menschen.—*Mitteilungen aus der med. Fakult Tokio*, 20, 345, 1918.
381. Salant, W., and Wise, L.: The Production of Glycosuria by Zinc Salts.—*Jour. of Biol. Chem.* 24, 447, 1918.
382. Salomon, M.: Geschichte der Glycosurie von Hippocrates bis zum Anfange des 19 Jahrhunderts.—*Deutsch Arch. für klin. Med.* 8, 489, 1871.
383. Salomon, H.: Ueber den Diabetes innocens der—Jugendlichen zugleich ein Beitrag zur Frage des renalen Diabetes.—*Deutsch. med. Wochenschr.* 40, 217, 1914.
384. Sansum, W., and Woodyatt, R.: Studies on the Theory of Diabetes VIII. Timed Intravenous Injections of Glucose at Lower Rates.—*Jour. of Biol. Chem.* 30, 155, 1917.

385. Scadding, H.: Report of Work of the Committee of Actuaries and Medical Directors. An Endeavor on the Part of the Canadian Companies to Secure Greater Uniformity in the Treatment of Impaired Risks.—Trans. of the 33rd Annual Meeting of the Assoc. of Life Ins. Med. Dir. 1922, 229.

386. Schenk, F.: Ueber Bestimmung und Umsetzung des Blutzuckers.—Arch. für die gesamt. Physiol. 55, 203, 1894.

387. Schneiderman, H.: Renal Glycosuria.—Jour. of the Amer. Med. Assoc. 80, 825, 1923.

388. Schöndorff, B.: Untersuchungen über die Ausscheidung von Zuckers im Harn von gesunden Menschen nebst einer Methode der quantitativen Bestimmung kleiner Zuckermengen im Harn.—Arch. für die gesamt. Physiol. 121, 572, 1908.

389. Schröder: Stoffwechsel der Kaninchen bei Quecksilbervergiftung.—Diss. Wurzburg, 1893.

390. Schülze, E.: Ueber Störungen des Kohlenhydratstoffwechsels bei Geisteskranken.—Verhandl. dei Gesellsch. deutsch. Naturforsches und Aerzte. Köln 1908, II 358.

391. Schülze, E., and Knaue, A.: Störungen des Kohlenhydratstoffwechsels bei Geisteskranken.—Allgem. Zeitschr. für Psychiatrie 66, 759, 1909.

392. Scott, E.: The Content of Sugar in the Blood Under Common Laboratory Conditions.—Amer. Jour. of Physiol. 34, 271, 1914.

393. Seegen, J.: Zucker im Blute, seine Quelle und seine Bedeutung.—Arch. für die gesamt. Physiol. 34, 388, 1884.

394. Seegen, J.: Ueber die Bedeutung und über den Nachweis von kleinen Mengen Zucker im Harn.—Wiener klin. Wochenschr. 1892, pp. 42, 95, 115, 127.

395. Seelig, A.: Ueber Atherglykosurie und ihre Beeinflussung durch intravenöse Sauerstoffinfusionen.—Arch. für exp. Path. und Pharmak. 52, 481, 1905.

396. Seelig, A.: Ueber den Einfluss der Nahrung auf der Aetherglykosurie.—Arch. für exp. Path. und Pharmak. 54, 206, 1906.

397. Seuringhaus, L., Kirk, E., and Heath, H.: Duration and Magnitude of the Hypoglycemia After Insulin.—Amer. Jour. of Med. Sciences 166, 677, 1923.

398. Shaffer, P.: On the Determination of Sugar in Blood.—Jour. of Biol. Chem. 19, 285, 1914.

399. Shaffer, P.: On the Normal Level of Blood Sugar of the Dog.—Jour. of Biol. Chem. 19, 297, 1914.

400. Shaffer, P., and Hartmann, A.: Iodometric Determination of Copper and Its Use in Sugar Analysis. Methods for the Determination of Reducing Sugars in Blood, Urine, Milk and Other Solutions.—Jour. of Biol. Chem. 45, 365, 1920-21.

401. Sherrill, J.: The Diagnosis of Latent or Incipient Diabetes.—Jour. of the Amer. Med. Assoc. 77, 1779, 1921.

402. Siebke: Beitrag zur Frage des Nierendiabetes.—Deutsch. med. Wochenschr. 36, 1031, 1910.

403. Siegmund, P.: Beitrag zur Lehre des Urinveränderungen bei Geisteskrankheiten insbesondere bei der progressive Paralyse der Irren.—Allgem. Zeitschr. für Psychiatrie 51, 602, 1894-5.

404. Silberstein, F.: Warmeregulation und Zuckerstoffwechsel.—Verh. der Kongr. für inn. Med. 30, 93, 1913.

405. Spencer, J.: Some Observations on Sugar Tolerance with

Special Reference to Variations Found at Different Ages.—
Quart. Jour. of Med. 14, 314, 1920-21.

406. Staub, H.: Untersuchungen über den Zuckerstoffwechsel des Menschen. I. Mitteilung. Ueber das Verhalten des Blutzucker nach peroraler Zufuhr kleiner Glukosemengen.—Zeitschr. für klin. Med. 91, 44, 1921.

407. Straus, H.: Zur Lehre von der neurogenen und der thyreogenen Glykosurie.—Deutsch. med. Wochenschr. 23, 275 and 309, 1897.

408. Strouse, S.: Some Observations in Normal Blood Sugar.—Arch. of Int. Med. 26, 751, 1920.

409. Strouse, S.: Observations on Alimentary Hyperglycemia.—Arch. of Int. Med. 26, 759, 1920.

410. Strouse, S.: Renal Glycosuria.—Arch. of Int. Med. 26, 768, 1920.

411. Strouse, S., and Beifeld, A.: A Case of So-called "Renal Diabetes," Possibly Traumatic in Origin.—Jour. of the Amer. Med. Assoc. 62, 1301, 1914.

412. Stütz, L.: Ueber den Einfluss von Körperarbeit und Überwärzung auf die Zuckeraussimilationsgrenze eines gesunder Menschen.—Deutsch. med. Wochenschr. 1909, 2, 2023.

413. Sweaney, H., and Johnson, E.: An Investigation on the Picramic acid Method for Blood Sugar.—Jour. of Lab. and Clin. 8, 506, 1922-23.

414. Symonds, B. (Chief Medical Director of the Mutual Life Ins. Co. of N. Y.): The Blood Pressure of Healthy Men and Women.—Transactions of the 33rd Annual Meeting of the Assoc. of Life Ins. Medical Directors, 1922, 22.

415. Tachau, H.: Ueber alimentare Hyperglykämie.—Deutsch. Arch. für klin. Med. 104, 437, 1911.

416. Tachau, H.: Beitrag zum Studium des Nierendiabetes.—Deutsch. Arch. für klin. Med. 104, 448, 1911.

417. Tatum, A., and Cutting, R.: The Action of Quinin on Sugar Mobilization and its Bearing on the Question of Glycogenolysis.—Jour. of Pharmac. and Exp. Therap. 20, 393, 1923.

418. Taylor, A., and Hulton, F.: The Limit of Assimilation of Glucose.—Jour. of Biol. Chem. 25, 173, 1916.

419. Thalheimer, W.: The Relationship of High Blood Sugar to Furunculosis.—Jour. of the Amer. Med. Assoc. 76, 295, 1921.

420. Thalheimer, W., and Updegraff, H.: A Comparison of Several Clinical Quantitative Blood Sugar Methods.—Jour. of the Amer. Med. Assoc. 78, 1383, 1922.

421. Thannhauser, S., und Jenke, M.: Ueber das Verhalten der β -Glykose im menschlichen Organismus und über die Natur der im Serum gelösten Glykose.—Münch. med. Wochenschr. 71, 196, 1924.

422. Thannhauser, S., und Pfitzer, H.: Ueber experimentelle Hyperglykämie beim Menschen durch intravenöser Zuckeringection.—Münch. med. Wochenschr. 9, 2155, 1913.

423. Von Tilerti, N.: Nouvelles recherches expérimentales.—Arch. ital. de biol. 51, 123, 1909.

424. Titus, P., and Givens, M.: Intravenous Injections of Glucose in Toxemia of Pregnancy.—Jour. of the Amer. Med. Assoc. 78, 92, 1922.

425. Traugott, C.: Ueber alimentare Hyperglykämie und Glykosurie mit besonderer Berücksichtigung der innocenten Glykosurie.—Klin. Wochenschr. 1, 2384, 1922.

226 Thirty-Fifth Annual Meeting

426. Trommer: Untersuchungen von Gummi, Dextrin, Traubenzucker und Rohrzucker.—Ann. der Chem. und Pharm. 39, 360, 1841.
427. Tuchen, H.: Ueber die Anwesenheit des Zuckers im normalen Harn.—Arch. für path. Anat. 25, 266, 1862.
428. Underhill, F.: Do Hydrazine Derivatives Show the Typical Hydrazine Effect upon Blood Sugar Content?—Jour. of Biol. Chem. 17, 295, 1914.
429. Underhill, F.: The role of Calcium in the Regulation of Blood Sugar Content.—Jour. of Biol. Chem. 25, 447, 1916.
430. Underhill, F.: The Influence of Sodium Carbonate upon Blood Sugar Content and upon Epinephrin Hyperglycemia and Glycosuria.—Jour. of Biol. Chem. 25, 463, 1916.
431. Underhill, F., and Baumann, E.: The Influence of Alkali upon the Creatinuria of Phlorhizin Glycosuria.—Jour. of Biol. Chem. 27, 147, 1916.
432. Underhill, F., and Baumann, E.: The Interrelations of Blood Fat and Blood Sugar Content of Dogs Under the Influence of Hydrazine.—Jour. of Biol. Chem. 27, 169, 1916.
433. Underhill, F., and Blatherwick, N.: The Influence of Subcutaneous Injections of Dextrose and of Calcium Lactate upon the Blood Sugar Content and upon Tetany After Thyreoparathyroidectomy.—Jour. of Biol. Chem. 19, 119, 1914.
434. Underhill, F., and Blatherwick, N.: The Influence of Thyreoparathyroidectomy upon the Sugar Content of the Blood and the Glycogen Content of the Liver.—Jour. of Biol. Chem. 18, 87, 1914.
435. Underhill, F., and Clossen, O.: The Mechanism of Salt Glycosuria.—Amer. Jour. of Physiol. 15, 321, 1905-06.
436. Underhill, F., and Kleiner, I.: Further Experiments on the Mechanism of Salt Glycosuria.—Jour. of Biol. Chem. 4, 395, 1908.
437. Underhill, F., and Wileus, G.: Studies in Carbohydrate Metabolism XXI. The Relation of Sugar Concentrations to Renal Integrity.—Jour. of Biol. Chem. 58, 153, 1923.
438. Velut, A.: Beitrag zum experimentelle Studium von Nebennieren-Glykosurie.—Arch. für path. Anat. 184, 345, 1906.
439. Wacker, L.: Untersuchungen über den Kohlenhydratstoffwechsel I. Mitteilung. Eine kolorimetrische Blutzuckerbestimmungsmethode und deren Einwendung.—Zeitschr. für physiol. Chem. 67, 197, 1910.
440. Watanabe, C.: Influence of Injected Guanidine Hydrochloride upon Blood Sugar Content.—Jour. of Biol. Chem. 33, 253, 1918.
441. Watanabe, C.: The Influence of the Administration of Calcium upon Blood Sugar Content in Rabbits with Guanidine Hypoglycemia.—Jour. of Biol. Chem. 34, 73, 1918.
442. Watanabe, C.: The Change of Phosphate and Calcium Content in Serum in Guanidine Tetany and the Relation Between the Calcium Content and Sugar in the Blood.—Jour. of Biol. Chem. 36, 531, 1918.
443. Waymouth, E.: A Method for the Estimation of Sugar in Blood.—Jour. of Physiol. 20, 316, 1896.
444. Wedenski, N.: Zur Kenntniss der Kohlenhydrate in normalen Harn.—Zeitschr. für physiol. Chem. 13, 122, 1889.

445. Wehner, W.: Some Vagaries of Albumin Test.—Proceedings of the 24th Annual Meeting of the Life Ins. Medical Directors, 1913, 198.

446. Weigert, F.: Der Uebergang des Blutzuckers in verschiedene Körpersäfte.—Arch. für Physiol. 1891, 187.

447. Weiland, W.: Ueber den Einfluss ermüdender Muskelarbeit auf den Blutzuckergehalt.—Deutsch. Arch. für klin. Med. 92, 223, 1907-08.

448. Weiland, W.: Über einige atiologisch bemerkenswerte Diabetesformen.—Deutsch. Arch. für klin. Med. 102, 167, 1911.

449. Welz, A.: Physiologische Amylogene Hyperglykämie.—Arch. für exp. Path. und Pharmak. 73, 159, 1913.

450. Hewitt, J., and Pryde, J.: The Metabolism of Carbohydrate, Part I. Stereo-chemical Changes Undergone by Equilibrated Solutions of Reducing Sugars in the Alimentary Canal and in the Peritoneal Cavity.—Biochem. Jour. 14, 395, 1920.

451. Wilder, R., and Sansum, W.: d-Glucose Tolerance in Health and Disease.—Arch. of Int. Med. 19, 311, 1917.

452. Wilhelmj, C.: Sources of Error in the Epstein Method for Blood Sugar Determination and a Modified Technic.—Jour. of Lab. and Clin. Med. 7, 489, 1921-22.

453. Williams, J., and Humphreys, E.: Observations on Tolerance and Rate of Utilization of Glucose in a Series of Individuals Exhibiting Various Degrees of Diabetes Mellitus.—Arch. of Int. Med. 23, 559, 1919.

454. Williams, J., and Humphreys, E.: The Clinical Significance of Blood Sugar in Diabetes Mellitus.—Arch. of Int. Med. 23, 546, 1919.

455. Willis, T.: Pharmaceutice Rationalis Sine Diatriba de Medicamentorum Operationibus in Humano Corpore.—Oxford 1674, p. 164.

456. Willis, T.: Pharmaceutice Rationalis; or an Exercitation of the Operations of Medicines in Human Bodies.—London, 1684, p. 91.

457. Wilson, E.: Blood Sugar Tolerance as an Index in the Early Diagnosis and Roentgen Treatment of Hyperthyroidism.—Jour. of Lab. and Clin. Med. 5, 730, 1919-20.

458. Winter, L., and Smith, W.: On the Nature of the Sugar in the Blood.—Jour. of Physiol. 57, 100, 1923.

459. Winter, L., and Smith, W.: On the Effect of Adrenalin on the Nature of the Blood Sugar.—Jour. of Physiol. 57 Proceedings, p. LIII, 1923.

460. Wishart, M.: The Permeability of Blood Corpuscles to Sugar.—Jour. of Biol. Chem. 44, 563, 1920.

461. Withams, J., and Humphreys, E.: Clinical Significance of Blood Sugar in Nephritis and Other Diseases.—Arch. of Int. Med. 23, 537, 1919.

462. Wolf, A.: A Study of the More Common Methods Used to Detect Sugar in Urine, with Special Reference to the Tests Employed in the Laboratory of the Equitable Life Assurance Society.—Transactions of the 31st Annual Meeting of the Assoc. of Life Insurance Medical Directors, 1920, 316.

463. Wolnik, B.: Experimentelle Untersuchungen über das Adrenalin.—Arch. für path. Anat. 180, 225, 1905.

464. Woodyatt, R., and Sansum, W.: d-Glucose Tolerance in Health and Disease.—Arch. of Int. Med. 19, 311, 1917.

465. Woodyatt, R., Sansum, W., and Wilder, R.: Prolonged and Accurately Timed Intravenous Injection of Sugar.—*Jour. of Amer. Med. Assoc.* 65, 2067, 1915.
466. Worm-Müller: Die Ausscheidung des Zuckers im Harne des gesunden Menschen nach Genuss von Kohlenhydraten.—*Arch. für die gesamt. Physiol.* 34, 576, 1884.
467. Worm-Müller: Die Ausscheidung des Zucker im Harne Nach Genuss von Kohlenhydraten bei Diabetes Mellitus.—*Arch. für die gesamt. Physiol.* 36, 172, 1885.
468. Worms, M.: Etudes cliniques sur le diabète.—*Bull. de l'acad. de méd. de paris* 3 Sér Vol. 34, 109, 1895.
469. Zuelzer, G.: Zur Frage des Nebennierendiabetes.—*Berl. klin. Wochensch.* 38, 1209, 1901.
470. Zuntz, N.: Zur Kenntniss des Phlorizindiabetes.—*Arch. für Physiol.* 1895, 570.
471. Harris, D.: The Functional Inertia of Living Matter.—London, 1908.
472. Jantzen, F.: Personal Communication.—To be published.
473. Nash, T.: The Kidney Factor in Phlorhizin Diabetes.—*Jour. of Biol. Chem.* 51, 171, 1922.
474. Joslin, E.: The Diabetic Problem of Today.—*Jour. of the Amer. Med. Assoc.* 83, 727, 1924.
475. Benedict, S., and Folin, O.: Metropolitan Laboratories.—Proceedings of the 34th Annual Meeting of the Assoc. of Life Ins. Medical Directors of America. 1923, 110.
476. Huston, R.: Glycosuria. Its Importance in Life Insurance Selection.—Proceedings of the 34th Annual Meeting of the Assoc. of Life Ins. Medical Directors of America. 1923, 205.
477. Truitt: In Discussion of Huston's Paper on Glycosuria in the Transactions of the 34th Annual Meeting of the Assoc. of Life Ins. Medical Directors. 1923, 216.
478. McCloud: In Discussion of Huston's Paper on Glycosuria in Proceedings of the 34th Annual Meeting of the Assoc. of Life Ins. Medical Directors of America, 1923, 220.
479. Strathy: In Discussion of Huston's Paper on Glycosuria in Proceedings of the 34th Annual Meeting of the Assoc. of Life Insurance Medical Directors. 1923, 221.
480. Ogden in Discussion of Paper by Blakely: Urin Analysis: A Review of Ten Years' Experience of the New England Mutual.—Transactions of the 27th Annual Meeting of the Assoc. of Life Ins. Medical Directors. 1916, 435.
481. Denis, W., and Hume, H.: On the Nature of Blood Sugar.—*Jour. of Biol. Chem.* 60, 603, 1924.
482. Felsher, H., and Woodyatt, R.: Studies in the Theory of Diabetes. IX Sugar Excretion Curves in Dogs Under Intravenous Injection of Glucose at Lower Rates.—*Jour. of Biol. Chem.* 60, 736, 1924.
483. Bertrand, G.: Le dosage des sucres réducteurs.—*Bulletin de la société chimique.* 3rd sér. 35, 1285, 1906.
484. Graunt, J.: Natural and Political Observations mentioned in a following Index and made upon the Bills of Mortality.—London 2nd Ed. 1662, p. 58.
485. Halley, E.: An estimate of the Degrees of the Mortality of Mankind, drawn from curious Tables of the Births and Funerals at the City of Breslaw; with an attempt to ascertain the Price of Annuities upon Lives.—*Philosophical Transactions.* London 17, 596, 1693.

486. Thorburn, J.: Female Risks.—Transactions of the 9th Annual Meeting of the Association of Life Insurance Medical Directors. 1898, 188.
487. Davis, J.: The Experience of the Union Central Life Insurance Company with Female Risks.—Transactions of the 11th Annual Meeting of the Association of Life Insurance Medical Directors. 1900, 257.

Dr. Ward—Those of you who were at the meeting last year, I think, will agree with me that possibly the most profitable hour of that very interesting meeting was the time we spent with Dr. Benedict and Dr. Folin as they revealed to us the work that had been done in the realm of sugar and albumin. Those are two constantly present problems with every life insurance man, and it is a very great pleasure to know that both of these gentlemen are here this morning, and I think that this coming hour is going to be a very important hour in this convention. I am going to ask Dr. Benedict in speaking to us not to confine himself to a discussion of this paper, but to give to us from his wisdom anything that he thinks will be of help to us in this great problem of glycosuria, for I am sure he realizes that it is a problem with every life insurance man to know what to do with these cases where occasionally we find sugar in the urine, and the problems that develop around that situation. Gentlemen, I take pleasure in presenting Dr. Benedict.

Dr. Benedict—Mr. Chairman and Gentlemen—Any of you who are familiar with Dr. McCrudden's writings—and I think many of you are, more especially those who are familiar with his monograph on uric acid that came out a number of years ago, would not have been at all surprised at the nature of the presentation he has made in the printed paper for these "proceedings," and would not be at all surprised at the difficulty he had in cutting that down to a mere skeleton as he has done. Nevertheless, it is an admirable presentation. It is essentially and wholly non-critical. It gives everyone an opportunity to find all that has been done on the subject within any reasonable limits, and I think everyone owes a great

230 Thirty-Fifth Annual Meeting

debt to Dr. McCrudden for such presentation as he has made there. It represents a great amount of work and care that is almost inconceivable.

I shall not, of course, attempt to discuss any of the theoretical presentations made in Dr. McCrudden's paper, the amount of sugar in the blood, the action of insulin, and questions of that type which are not practical at the present time—which may be practicable tomorrow but are wholly impracticable at the present moment. I shall have to limit my discussion primarily to a few points that are especially fundamental in my opinion at the present time.

The first of those I shall mention very briefly, and it is apparently a point of very great importance—the question of the impairment of these risks showing the so-called "slight" glycosuria, which is reported. I should say that on the basis of past work, and the type of urine examinations that have been made, I do not believe the statistics are worth much, if anything. They have to be revised by the statistician, in view of the more accurate methods of analysis, before we can draw conclusions that the finding of sugar represents any specific result really. One man has done it one way and another man, another way, and you cannot include these cases as representing any specific group, unless the same man had analyzed the urines all the time.

I think, as a sort of text, I should perhaps take the statements quoted by Dr. McCrudden, and I shall dwell on one or two primary points.

Dr. McCrudden acquiesces in the statement made by Dr. Joslin a few years ago before this Association, that on the basis of qualitative urinalysis, any reaction found by the ordinary tests must be regarded as indicative of Diabetes Mellitus. I want to discuss that question from certain standpoints. First, of course, there is the question of what is meant by a slight glycosuria reaction. We shall take that up

later. Second, the question whether that statement is true. When one comes to disagree even at the opening of a discussion with Dr. Joslin, a man of his standing and ability in the field of diabetes, it seems to be a dangerous proposition, and naturally my disagreement with him is not as whole-hearted as it may appear to be in this discussion. But Dr. Joslin primarily presents the view of the man who sees diabetes day and night, year in and year out. The cases that come to him are, of course, mainly diabetic. There seems reason for the belief that they had diabetes, or at least for strongly suspecting it before he ever saw the cases. Therefore, most of the cases that show even a trivial reaction with him will have subjected themselves more or less to diet before they came to him. In other words, he does not meet the type of cases that the insurance man meets, who has presumably healthy men coming to him, nor the type of cases that I meet, who are presumably healthy men, men that I examine in the student's laboratories all the time. As an instance of that, during the years that I have been teaching in Cornell, we have made it a routine practice to examine qualitatively at any rate up to last year and since then quantitatively the urine of every student. We have found in that period of time some eleven students, who have given very strong positive reactions with Fehlings and with the ordinary copper solutions, and an additional group of perhaps fifteen gave slight reactions, but still enough to arouse suspicion. Not one of those students followed through a period of from one to three years after that reaction has developed Diabetes Mellitus. That brings up the question whether diabetes is a disease that develops slowly, or whether it is an acute condition, and I would say at the present time that it is an acute condition that does not develop through a long, slow, gradual loss or impairment in the sugar-burning power. We have some evidence that may be interesting. First I might cite some that Dr. Allen prepared for me a con-

siderable number of years ago, when he took out the pancreas of dogs in very small portions, and first delivered to me two dogs which he guaranteed, if they were fed up with sugar, would develop diabetes. I took those two dogs, put them in cages, followed the urine qualitatively very carefully, had no quantitative method, and I just poured glucose into those dogs, I put it under their skins and soaked them in it, and I couldn't faze their sugar combustion. It was just the same as that of a normal dog. Dr. Allen had left a pancreas the size of a twenty-five cent piece. They never developed diabetes nor even a border-line condition. They were perfectly normal. Dr. Allen guaranteed they would develop it, but they didn't. He came down to the laboratory later and operated some animals there, and I might mention that it is a marvellous sight to see Dr. Allen's technique. He would leave in small portions of the pancreas and then I endeavored to get these dogs diabetic. One or two of them that were introduced to glucose did develop diabetes, but the curve was not a gradual rise at all. Suddenly those animals would go all to pieces, with sugar pouring into the urine—no gradual transition period whatever.

Now, let us go over the recent results reported by Wood-yatt, in which he has introduced glucose continuously into a normal dog, and studied the output of sugar in the urine to see whether the tolerance breakdown is sudden or very gradual. He finds that the technique of the normal sugar curve is something like twenty milligrams per hour; with the introduction of small amounts of sugar that may rise to twenty-three or twenty-four milligrams per hour; with the introduction of still larger amounts, to twenty-five or twenty-six milligrams. The rise is very small, but there is a tendency for the curve to go up just a little bit, as he runs up his sugar. Then, this curve is almost a straight line to a certain point and at that point this curve goes up quickly, showing that the carbo-

hydrate tolerance of that animal is reached suddenly, and suddenly goes to pieces, and the sugar pours out. Is that a condition, going from the dog to the human, which ever obtains in the diabetic? It absolutely does, and the finding of it was the greatest surprise to me, but I believe it, and it has been corroborated by Woodyatt's pupils, by Myers and by myself. The condition is just this: Woodyatt starved the diabetic until he was sugar-free, and then he wanted to see whether this diabetic, being given a diet containing increasing amounts of carbo-hydrate would give a gradual curve, or would go to pieces suddenly. His results were absolutely clear-cut, that the diabetic was normal to a certain point, he gave the sugar in increasing amounts, there was no tendency to a slight glycosuria, but suddenly the urine filled up with 1 to 2% of sugar. I got the same reaction and so did Myers. That is why I feel that the interpretation of small traces of glycosuria has got to be that the individual is not a diabetic until you prove him so, unless the sugar runs up to a very considerable percentage. You are not from the standpoint of safety going to throw out a man with .2% of sugar, any more than to throw him out if he has only .1% of sugar. That is from the data available at present.

The final point in connection with Dr. McCrudden's paper presents the procedure of the New England Mutual, of accepting a case that shows what he calls a slight glycosuria. They make the blood test, and if the blood sugar within two hours rises to .12 per cent, by the Folin method, that case is on the border-line—if above that, reject, if below that, accept. So far as that blood sugar test control method is concerned, I agree with Dr. McCrudden absolutely that it is the best thing we have. It is what Dr. Joslin would do to determine whether a case has diabetes. It is the best thing that can be done if the suspicion is definitely raised, and you can get the individual to allow you to penetrate his vein and make

the test. But in connection with that decision of the New England Mutual's, and Dr. McCrudden's presentation of it, I do want to offer a very strenuous criticism of the situation there, in presenting at this time to this audience the qualification that if the case shows "a slight glycosuria." I feel that even at the present time that condition has been passed, that it should be on a definite basis, that Dr. McCrudden should tell us just what "a slight" glycosuria is, what makes him make those blood-sugar tests, whether .1, .2, .3, .4 or .6% of sugar. Dr. McCrudden has rather emphasized the use of Fehling's test, but if you pour in too much urine with a given volume of Fehling's, it gives too delicate, and if too little, vice versa and the whole thing should be gotten on the basis of knowing how much is there, and then the companies can without any difficulty interchange their results and know just exactly whether these cases do develop into diabetes where they show a very low percentage to begin with, but a little above what you and I might show at any given time. I should like to ask Dr. McCrudden from his clinical experience how many cases he has found to go into full Diabetes Mellitus or to actually have it where he has had to wonder on the basis of qualitative reaction whether that individual had sugar in the urine or not.

Dr. Ward—I am sure that we all want to hear more from Dr. Benedict, and we may have some questions to ask him.

We are all familiar with the work that has been done in this field in Toronto by Dr. Banting—in the great field of Diabetes. It was my great pleasure to see Dr. Banting a few weeks ago, and I hoped to have him with us today, but we have from Toronto, one who is familiar with Dr. Banting's work, Dr. Rolph, and I am going to ask him to speak at this time.

Dr. Rolph—Mr. President and Gentlemen—I am going to be very brief. In the first place, I have nothing but praise for

Dr. McCrudden's stupendous task which he has done so successfully, and I think that, as Dr. Benedict said, he has certainly given us the best method for differentiating between glycosuria as a symptom and Diabetes Mellitus, which we know, is a failure to function in the islands of the pancreas.

The question comes up, I think, as Dr. Benedict has said, can we accept cases that probably are really mild types. I think we are going to be able to take in as standard lives, but for short term policies, rated up, certain classes, and I think that the particular class that will come in, will be men well up to weight or overweight. The overweights, as a rule, certainly have a much better tolerance, between the ages of 30 and 50. There are a large number of those cases that show occasional glycosuria and must be classed as diabetics or potential diabetics. As a matter of fact, a large number of these cases are getting standard insurance. I have done quite a bit of diabetic work, and I have made it a practice to ask these people what about their insurance? Have they any? And it is astonishing, especially the overweights of a certain race that are particularly prone to diabetes, the number of them that have ten or fifteen thousand dollars insurance. Several of them have told me that the agent always examines their urine beforehand, and if he finds sugar, he tells them to starve for a few days, and then go and be examined. Just a couple of months ago, I had a case like that—a man I had known very well who had had three attacks of acute pancreatitis, and showed sugar every once in a while, and he had gotten \$15,000 standard insurance without any trouble. Another man who was a definite diabetic and absolutely uninsurable, had gotten \$10,000 insurance. There are a large number of those cases, particularly of a certain race, who are getting standard insurance, and I think we have got to be a little more particular in our overweights, whose blood pressure is perhaps up a little bit, and I make it a practice to drop around

236 Thirty-Fifth Annual Meeting

and get a sample of the urine when they are not expecting it, and occasionally, indeed, quite often, find that they have some sugar.

I had hoped to get some statistics from the Toronto General before Dr. Banting's discovery and since, but we have been so overcrowded with patients from all over the world, that there is no possibility of making the comparisons.

Our coma cases are good. For instance, we have a Chinaman who holds the long distance championship. He has been in nine times with coma, and I saw him in the hospital the other day recovering from his ninth attack, and he was writing on the blackboard—"pie," "cake," "candy"—so you can see what he was going to do when he gets out again. It is very interesting.

But I think the blood-sugar test is going to put on the safe side of the fence a fairly large number of renal cases, and I think we will have to distinguish between renal cases and toxic renal. There is the nephritic renal and the diabetic renal and a group we are recognizing and calling Diabetes Innocens, which is apparently between the two. I do not think we have the last word in the renal yet. I saw a case not long ago of a man very thoroughly investigated for a long time. Everything showed a very definite renal diabetes, but it just happened shortly after I had seen him that he had some trouble with the face, also some antrum trouble and later had a tooth pulled. The sugar entirely disappeared, but for six months afterward he used to come in every few weeks, and we were never able to get any sugar in his urine. That case can hardly be classed as a renal diabetic.

It is easy enough in the cities perhaps to get tests of the blood, but one point I would like to make is that if we are doing Dr. Benedict's test, let us do his test as he directs it should be done, and not as anybody thinks it might be done. You will find the directions on the bottles distributed by whole-

sale houses who send out Benedict's solution, as to how to do the test, and they say, take about 5ccs. and put in eight or ten drops of urine, and boil it for a minute or two. If you get a precipitate, you have probably got sugar. Now, if you are going to do a man's test, do it as he directs and as he says. I have been around the country, in small towns, and I have talked to the Ontario Medical Association, and I have seen them trying to do tests. I remember in Fort Hope, five men were having a debate as to the various tests, and not a man there had the faintest idea of doing the tests right. We find the same with students, after very careful instruction, extending over a whole term, they will make a test for sugar, and put 5 ccs. of urine to 10 drops of Benedict's solution, and get positive results. I think it is very important for us to insist on following the directions and to write our Examiners occasionally giving them complete details of these tests, and try to get them to do the tests as they should be done.

The President asked those members present who are using more or less the blood chemistry in this problem to raise their hands. A comparatively small number did so.

Dr. Ward—Dr. Bradshaw, may we hear from you?

Dr. Bradshaw—Mr. President and Members of the Association—I think Dr. Benedict's suggestion is an excellent one, to determine before we go into the blood chemistry part of the problem, what type of cases blood chemistry should be applied to. The ones that we see and the ones on which we request blood chemistry to be done are the ones in which we really get a definite amount of sugar in the urine, a half per cent or more. You can test them out in various ways. The agents have learned how to get them sugar-free, and when we have found a definite amount, and the question of the amount of insurance comes up, I think that is the only method by which we can catch them. Dr. McCrudden's suggestion as to how to do it apparently is absolutely sound. I have talked to several men who do blood work, and they all

238 Thirty-Fifth Annual Meeting

agree that the suggestion would catch the diabetic. The criticism I have of it is that the test must be made by a man who knows how to do it. I do not believe that throughout the country there are very many laboratories that would give us very accurate results. Another thing that occurs to me is, how long after getting the blood must the test be made? The clinical pathologists say that certain changes take place about an hour after the blood is drawn, that sugar is changed to higher forms, and that we do not get accurate percentages. If this is so, how are we going to get blood from a man who lives in one of the smaller towns to a laboratory? The test, it seems to me, will have to be adopted in questionable cases, where the insurance amounts to something.

Dr. Ward—Dr. Dwight, you are one of those who handle this question. May we hear from you?

Dr. Dwight—Mr. President and Gentlemen of the Association—Perhaps I can clear up some of the questions which I see are being asked, both by Dr. Benedict and some of the other gentlemen, a little better than Dr. McCrudden, who really did the work. I don't know whether I am to pose as the devil or the deep sea. I think that probably Dr. McCrudden thinks of the first gentlemen in connection with me, and I don't blame him, because in asking him to make this study I was trying him out, and I think that those who will read that paper carefully will agree with practically what Dr. Benedict has to say, that he has done that job in the most wonderful way. I got all that I expected and a great deal more.

Now, as to Dr. Benedict's question, what class of cases are we testing? Well, the greatest problem that we have at the Home Offices of our Companies is to decide what to do with those people who come to us with a definite history of an impairment, whether it is sugar or albumin or heart or anything else, coming from an unknown source, we knowing nothing about the individual who made the test, nothing

about his ability or the character of the test which he used, but standing before us is the record, sugar, albumin, casts, heart murmurs, hypertrophy, etc. We cannot run away from it. Every man who has done any statistical work on life insurance cases will agree absolutely with Dr. Rogers and Mr. Hunter in the figures which they furnish, and it is extraordinary how accurately or how absolutely the figures of the New England Mutual agree with theirs. They are honest figures, taken in the same way, because I learned the method from Dr. Rogers, and they work. There may be a variation of one or two points in mortality. That is about all. We are faced, Dr. Benedict, with this question. We find that an applicant for insurance who perhaps passes a perfectly normal examination when he comes to us has a history of having had sugar in the urine. This is all we know. We know that if we accept a thousand of those risks, we will have a mortality of approximately 180 points of the American Table—it may be 150, it may be 120. We cannot afford to take those risks as they then stand. You gentlemen who are doing substandard insurance—and most of you are in one way or another—have a way of dealing with them which I will not go into—we hope not to go into it—we don't want to—we believe in it, it is all right, but we would rather not. Therefore we must have a way of sorting approximately and with sufficient accuracy for our purposes that one big group into two groups, one of which will give us a comparatively normal mortality. In our Company many years ago, we adopted the method of separating a group of albumin and casts, which was a very large group, into two groups, by applying a load to the kidney, a measure load, by the specific gravity and the percentage of urea, and we said, if a man, being perfectly normal in every other way, can pass a urine with that specific gravity and that urea, absolutely normal in other ways, when examined by skillful men, he is sufficiently normal for our purposes. That was not science. It was a working hypothesis which

240 Thirty-Fifth Annual Meeting

we have tried out for twenty years with great success, so far as we are concerned. We are perfectly satisfied. The mortality of a group accepted in that way is less than the average mortality of our whole business taken during the same period. It may not be scientific, it may not be accurate, but it works, and that is my job—to make it work. For many years we have been trying to find a similar method of handling another very large group—circulatory disturbances—in hearts and blood pressures &c. We have evolved a way by which we can apply a measure load and can see the measured result. We are basing our decisions in those doubtful cases on the same lines. So when Dr. McCrudden came with us, shortly after he had begun to get our point of view, I asked him to do two things—I had no time and did not have the intelligence or the ability to take up such a subject and go through with it—I asked him to tackle it because I believed he was the man—to give me, so that I could have in my desk—what there is on the subject, and also suggest to me a load which we can apply to this great heterogeneous class of all kinds of things—fakes and not fakes—Jews and Gentiles—who are said to have had sugar. We have that test and we are applying it because it seemed reasonably in accord with our previous experiences on other classes. Now, there are certain difficulties in applying it that have been referred to. Dr. Rogers asked me the other day if we were prepared to defend suit by some of these people where we did blood sugars. We are trying to avoid such suits by putting it up to the applicant to furnish us with a blood sugar tolerance test made by a specialist whose report would be satisfactory to our chief examiner in that particular city. He can go to anybody he pleases. If he has been under treatment by a specialist he can furnish us with a report on blood sugar which has already been made, provided it is within a reasonable time. If he doesn't know any man he cares to go to, we will do it at his request. If he

prefers his own doctor, he pays for it; if we do it at his request, it is free of charge. We have here a great heterogeneous class. We try to control the men who are making the examinations, and we are trying out this method not with the idea that it is the ultimate thing, but in the hope that it will work as our other loads have worked, and give us the ability to pick from this great mass of applicants a large group who are safely insurable at ordinary rates. I cannot give you the exact figures, but my impression is, that we are accepting from 50 to 60% of those people. Well, this is a saving, because I have learned by experience that I have no other way of picking them which gave me anything below 120 to 150 and 180 points of the American Table. We have only been doing it a short time, and we are just trying it out, but I feel glad I am able today to give every Company connected with this Association the benefit of all the information which I knew I needed in the best form I have ever seen it to lay on their desks. No man can do such a piece of work and not have it open to some criticism and some doubt. There are no two men in this room who will agree, but I believe they have got what I know I need.

Dr. Ward—It is a very great pleasure, gentlemen, for me to present to you Dr. Folin, who is going to say something on this subject.

Dr. Folin—Mr. President and Gentlemen—I have not read Dr. McCrudden's paper, and I came in late and did not realize the importance of this discussion. I want to begin, however, by saying that I have known Dr. McCrudden for twenty years, and he used to be a very good man, but like other good men, he seems to me to be sliding down. I think that is too bad. The main point that I want to raise this morning is that it is unfortunate that a man of Dr. McCrudden's experience comes here and talks that old tale to you. That is, I think, retrogression. If we want to use qualitative methods, why not use Fehling's? I don't use Fehling's except to show

that it is no good, and I invariably tell my students that it is no good, and now Dr. McCrudden comes into an insurance company that falls back and uses this method, and he tells us that that is the thing to do. Well, Dr. McCrudden, you shouldn't have done that. There is no progress in that. What in the world are we men doing if we can't do any better than what was done twenty-five years ago? You give us twenty-five years of the quantitative method, and I think you will see something. It seems to me it is silly for a group of men dealing with important problems as you are not to take note of the advances that are being made, but rather slip down, by clinging to practices that were justifiable when you were young. Statistical material based on scientific material cannot be obtained by those methods.

Dr. Ward—Has anyone else anything to contribute on the subject of blood-sugar chemistry? We will be glad to have another word on that subject. Dr. Rockwell, are you using this method at all?

Dr. Rockwell—We are following very largely the thought Dr. Dwight brought out. We are not using it extensively or as a matter of routine. We are equipped in our laboratory to make blood tests. We make them there sometimes, but we would very much prefer at this juncture to be supplied with the results of that blood test by some other laboratory than our own. Evidently Dr. Rogers and Dr. Dwight have talked this matter over from the standpoint of possible suit. I think it is a pretty good rule for all life insurance companies to adopt not to permit in the examination of their applicants any instrumental methods. We have run up against some difficult points that way. We instruct our examiners not to use any instrumental methods whatever, and if they do, we could show the Court that they were acting contrary to our definite instructions. It is largely this way with blood sugar tests. We welcome reports from good laboratories, and we get them, and there are cases where we put it up to the indi-

vidual to furnish us with them one way or another. I believe it is something that is coming—that we must all take cognizance of.

Dr. Ward—Has Dr. Patton anything to add to this subject?

Dr. Patton—I have nothing to add, Mr. President, to what Dr. Dwight and Dr. Rockwell have said. We are not equipped, as Dr. Rockwell is, in our own laboratory, but we have had blood sugar tests made on specimens sent in, or we have accepted occasional reports from well-known laboratories recommended to us by our chief examiner in the locality. We do not pay for the blood sugar test. We put it up to the applicant that if he is willing to furnish to us the results of such a test, we will give his case that much more thorough or careful consideration.

Dr. Ward—Dr. Benedict, have you a closing word for us?

Dr. Benedict—Mr. Chairman—There are just one or two points that have been brought out. In the first place, I want to say again that the idea that you can catch the diabetic who has the situation in hand by looking for a trace of sugar just above the normal is, until it proves to be the contrary, absolutely erroneous. That individual by fasting will get just as low as you or I at our best, and you won't catch him that way. That is my opinion, after trying the thing repeatedly, up to the present time. If insurance statistics will show otherwise, we will of course revise that viewpoint.

With regard to the question of blood sugar determination, one thing has come out during the past year that is going to make it a little more practicable, and that is the finding by a Wisconsin laboratory that fluorid and thymol added to the blood will absolutely prevent the decomposition of the sugar for a long period of time, so that it can be shipped and apparently with perfect safety over the country from any point a man might wish to supply it.

The points brought out in the procedure of the New Eng-

land Mutual have not answered the questions I have brought up, because they base their blood sugar reports on the report that the individual has at some time had sugar. Now, of course if he had sugar, you have got to make some very careful test. He may have had a little sugar or he may have had a great deal of sugar. Now, the point we want to get at is, how many cases showing just above the normal levels do on that test develop true diabetes, and I wonder if Dr. McCrudden has any data on it?

Dr. McCrudden—I think I will have to take my cue from Dr. Benedict in the admirable self-restraint he has shown in discussing the paper and leaving out the things we might talk about, in view of the time I should take. I know what he has in mind and I should like to discuss it, but it would carry me too far to make an answer satisfactory to him now. I thank Dr. Benedict, and the other gentlemen who have so kindly discussed the paper.

Dr. Ward—We will now have one of the most important features of the convention, and that is, the report to be submitted by Dr. Patton with relation to some features of the albuminuria problem, and then the privilege of having some of our chemists to discuss that problem.

Dr. J. Allen Patton presented the following report:

COMMITTEE ON URINARY IMPAIRMENTS.

The Association of Life Insurance Medical Directors adopted the report of a Special Committee on Urinalysis at their Twenty-Eighth Annual Meeting in 1917, and most of the companies have reported their impairments accordingly. The Heller's nitric acid test is qualitative and many companies now depend upon quantitative methods in their doubtful cases.

At their May, 1924, meeting, after considering correspondence concerning the comparative results obtained by Heller's and Purdy's tests, the Executive Council approved the fol-

Committee on Urinary Impairments 245

lowing motion: "That the President appoint a committee to receive reports from chemists and others working along the lines of albumin determination in life insurance work and to correlate them and report on the subject of reporting urinary impairments at the next meeting of the Association." President Ward appointed Drs. J. Allen Patton, A. S. Knight and T. H. Rockwell as the committee.

The committee has met several times, having Drs. Clark, Exton, Folin, Kingsbury, Rose and Wolf at some of the meetings. The laboratories of the Equitable, Metropolitan, Mutual Benefit and Prudential have been in constant touch in this albumin work and a number of other insurance laboratories have been requested to consider that subject.

The committee was unanimously of the opinion that the urinalysis methods recommended by the Association should be revised and that quantitative albumin tests should be substituted for qualitative, the reports made in percentages and limits be established for N, M and L amounts.

We therefore recommend:

1. The Association members be urged to use quantitative tests and report accordingly.
2. The following limits for N, M and L records:

| | | % | Mille % | Heller's Test |
|---|----------|-----------|----------|-------------------|
| N | Small | .01 - .05 | 10- 50 | Faint trace—trace |
| M | Moderate | .051-0.1 | 51-100 | Moderate |
| L | Large | Over 0.1 | Over 100 | Large |

Less than .01% or 10 mgm. per 100 c. c. urine should not be reported as an impairment.

3. Sulphosalicylic acid at the present time has advantages over the other protein precipitants used for detecting albumin in urine and, therefore, it is recommended as the basic reagent and optional methods as described in the report of Drs. Kingsbury and Rose, are suggested for use during the coming year.

MEMORANDUM FOR DR. J. ALLEN PATTON,
CHAIRMAN.

The subcommittee of chemists, Drs. Kingsbury and Rose, have made 57 protein determinations on specimens of urine in the Metropolitan Bio-Chemical Laboratory, which have been officially reported as containing 20 or more mgms. protein per 100cc, these determinations of urine made by three different procedures, all based on the precipitation of protein by sulphosalicylic acid.

A—*Reagent*: 2% sulphosalicylic acid.

Dilution: 1 volume urine—24 volumes reagent.

Standard: Known concentrations of sheep serum with 15% salt solution made up at the same time and in the same manner as for the precipitation of the unknown.

B—*Reagent*: 5% sulphosalicylic acid—1% sodium chloride—20 c. c. saturated aqueous solution of brom phenol blue per liter.

Dilution: 1 volume reagent—1 volume urine.

Standard: Sealed tubes containing known amounts of protein precipitated by the reagent in a manner identical with the determination, except as to length of time of standing.

C—*Reagent*: 3% sulphosalicylic acid.

Dilution: 7.5 volumes reagent—2.5 volumes urine.

Standard: Protein sulphosalicylate suspended in 10% gelatin (firm gel) as a protective colloid, and standardized against known sheep serum. Solution precipitated in a manner similar to that used with urine.

In these tests all readings were made at the end of ten minutes.

The data are tabulated in the accompanying table. The first column identifies the specimen; the subsequent three columns are the values obtained by the respective turbidimetric

Committee on Urinary Impairments 247

methods. When there was a discrepancy between A, B and C your subcommittee made a gravimetric determination of the protein and the results are given in Column D.

It will be noted that, in general, the three methods are within reasonable agreement. B gives slightly lower values, and A slightly higher, but the variations are not significant in life insurance procedure. Of all 57 specimens, only two differed sufficiently to justify gravimetric determinations.

| Sample | Optional | | | | Gravimetric | | | | Sample | Optional | | | | Gravimetric | | | |
|--------|----------|-------|------|-----|-------------|-----|-----|----|--------|----------|-------|-----|-----|-------------|---|---|---|
| | A | B | C | D | A | B | C | D | | A | B | C | D | A | B | C | D |
| 652 | 50 | 30+ | 40 | — | 447 | 50+ | 40+ | 50 | — | 524 | 125 | 60 | 100 | 77 | — | — | — |
| 893 | 65 | 60 | 60 | — | — | — | — | — | 633 | 1200 | 1000 | 200 | ++ | — | — | — | — |
| 845 | 75 | 50-60 | 60 | — | — | — | — | — | 640 | 50 | 30+ | 35 | — | — | — | — | — |
| 831 | 180 | 135 | 150 | 105 | — | — | — | — | 638 | 170 | 150 | 150 | — | — | — | — | — |
| 791 | 40 | 30 | 20 | — | — | — | — | — | 625 | 75 | 50 | 50 | — | — | — | — | — |
| 787 | 750 | 500+ | 650 | — | — | — | — | — | 641 | 50+ | 50 | 45 | — | — | — | — | — |
| 763 | 200 | 150 | 150 | — | — | — | — | — | 648 | 40 | 30-40 | 30 | — | — | — | — | — |
| 914 | 50 | 40- | 30 | — | — | — | — | — | 694 | 40- | 30 | 30 | — | — | — | — | — |
| 938 | 80 | 60 | 60 | — | — | — | — | — | 698 | 40 | 25 | 30 | — | — | — | — | — |
| 936 | 80 | 80 | 80 | — | — | — | — | — | 671 | 100 | 90 | 75+ | — | — | — | — | — |
| 942 | 45 | 30-40 | 40 | — | — | — | — | — | 677 | 300 | 200± | 300 | — | — | — | — | — |
| 907 | 60 | 50+ | 55 | — | — | — | — | — | 687 | 50- | 30+ | 30+ | — | — | — | — | — |
| 881 | 125 | 100 | 100+ | — | — | — | — | — | 5 | 100 | 80 | 100 | — | — | — | — | — |
| 844 | 80 | 50 | 75- | — | — | — | — | — | 16 | 50 | 40 | 40 | — | — | — | — | — |
| 811 | 40 | 25 | 20 | — | — | — | — | — | 81 | 35 | 20 | 10- | — | — | — | — | — |
| 23 | 80 | 60 | 60 | — | — | — | — | — | 84 | 40 | 30+ | 40 | — | — | — | — | — |
| 30 | 200+ | 320 | 350 | — | — | — | — | — | 56 | 85 | 90 | 85 | — | — | — | — | — |
| 973 | 125 | 100 | 100 | — | — | — | — | — | 76 | 75 | 60+ | 70 | — | — | — | — | — |
| 957 | 200 | 225 | — | — | — | — | — | — | 111 | 100+ | 100+ | 100 | — | — | — | — | — |
| 959 | 40 | 40 | 40 | — | — | — | — | — | 114 | 60 | 40 | 50 | — | — | — | — | — |
| 981 | 40 | 30 | 35 | — | — | — | — | — | 983 | 40 | 30 | 40 | — | — | — | — | — |
| 983 | 45 | 30 | 40 | — | — | — | — | — | 38 | 20 | 20 | 20 | — | — | — | — | — |
| 596 | 50+ | 40-40 | 40 | — | — | — | — | — | 981 | 40 | 30 | 40 | — | — | — | — | — |
| 444 | 75- | 60 | 75 | — | — | — | — | — | 964 | 20 | 25 | 20 | — | — | — | — | — |
| 527 | 50+ | 40-50 | 60 | — | — | — | — | — | 990 | 35 | 30 | 20 | — | — | — | — | — |
| 534 | 200- | 180- | 100+ | — | — | — | — | — | — | — | — | — | — | — | — | — | — |
| 448 | 50 | 25+ | 30 | — | — | — | — | — | — | — | — | — | — | — | — | — | — |

Respectfully submitted,

COMMITTEE:

DR. J. ALLEN PATTON, Chairman.

DR. A. S. KNIGHT,

DR. T. H. ROCKWELL,

DR. WM. H. WARD,

Ex Officio.

Dr. Patton—I would like to comment on one or two thoughts in connection with this report. We found that there were some variations in the methods used in the preparation of the re-agent, and considerable discussion has been held on the question. The method reported as Method C, is one that was developed by Dr. Clark and Dr. Wolf, and those representing the Metropolitan laboratories. They did considerable work on that subject and they have developed what is believed to be a very reliable and good test for any of us to use. We are all agreed—and I do not believe we are going to get any disagreement on this statement—that we have not settled absolutely on standards for comparison, but we are also agreed that we have standards that are usable for life insurance purposes at the present time, and that is why your Committee is reporting these three methods for use during the coming year, with the idea of a further crystallization upon the subject.

The Prudential prepared and sent out some ten or twelve sets of standards. Unfortunately we found soon after they had gone out, that there had been some errors in their preparation, and that there was a fault in the reagent, and the reagent reported as Reagent B is the one we are now using in our laboratory with the corrections made in that. We have many discrepancies in our own laboratory. Undoubtedly some of you men who have used it in your laboratories have obtained discrepancies and if you have not something has been the matter with your technique. So that we are still in a manner of coming together on this subject. The hurried compilation of this report has prevented us in having ready for the Association today an outline of the preparation of these standards and the methods of their use. It is the intention to furnish each Company with such an outline just as soon as it can be prepared. It is not necessary to send out an outline of Method A, because that was covered in last year's "Proceedings," but it may be repeated in order to

bring all the methods together, so that they will be definitely set forth in one little pamphlet and will be accessible for ready use.

This Committee therefore as appointed was primarily for the discussion of Albumin, and possibly the members of the Committee would be willing to be discharged, but there is no question from the discussion we have had here this morning with regard to sugar that the door should not be closed on further work on urinary determination and urinary standards—not necessarily on compulsory standards—I do not believe this Association can tell you or me what we shall use in our laboratories. They can tell us what should be the basis for our reports to other Companies. They cannot tell us what we shall use for our own individual needs. For the good of the business, however, they should lay down certain lines or limits, within which we should make our reports for the common use of the Companies.

Dr. Ward—Only those who have been in close touch with this work have any conception whatever of the amount of labor expended upon it under the direction of this Committee. It has not been an overnight job, but has taken days and weeks and months of diligent and constant toil to get as far as the Committee has gotten. Now, I am going to ask Dr. Folin to speak to us on the work that has been done by the chemists, and to give us any recommendations he may have to make.

Dr. Folin—Mr. Chairman—I have nothing to add. It seems to me the report covers the situation perfectly well, and I think nobody could find any fault with it, and therefore it seems to me perhaps useless to waste your time with any detailed discussion of this sort especially if the methods are to be given to you in print. We have undoubtedly given much time to this work, and there was a time when we felt like pulling each other's hair, figuratively speaking, but we have come to substantially the same method.

I might call your attention to the fact that the only difference is that in one of the three methods there is a coloring matter, whereas in the other two methods it is straight sulphosalicylic acid. That coloring matter goes with the solution—where the color of the urine itself may cut some figure it may introduce some difficulty, in otherwise perhaps making your reading, but if anybody wants to use that solution and wants to have a coloring matter to match the color of the urine, there is no real objection to it by taking less urine. The need for an indicator becomes superfluous and it is really a matter of choice, a small detail which is not going to affect you one way or another. I would only caution you that when it comes to the question of permanent standards that problem is not yet solved. Please have that clearly in your minds. The permanent standard of the sort that can be used in all sorts of laboratories has not yet been found, and it is inevitable that it is going to take months of time, whether months of work or not, to determine whether any given standard is dependable.

Dr. Ward—Those who were at the meeting last year remember the demonstration Dr. Folin gave of the use of this sulphosalicylic acid test, and we are going to continue that at this time. Dr. Clark and Dr. Kingsbury are to demonstrate this to us:

DEMONSTRATION BY DR. CHARLES P. CLARK.

We wish to demonstrate that the quantitative test which we have devised is simple in execution. After much experimentation, those of our committee who have combined in developing this method, agreed to use $2\frac{1}{2}$ cc. of clear urine mixed with $7\frac{1}{2}$ cc. of a 3% sulphosalicylic acid solution. The test tube is then inverted, once or twice, and read, after ten minutes against our standards. This dilution insures a very definite turbidity when .01% albumin is present and, as seen by the set of standards, there is a very definite gradation

in the turbidity up to .1%. This dilution of 1 to 4 also very largely removes the difference in color between the standards and tested urine. Our experience in placing this in hands of technicians of ordinary education, indicates that essentially accurate readings will be obtained. and, as you must agree, no quantitative test could be more simple in execution.

A second point which we wish to bring out, is that we will not become more severe in dealing with our applicants. In fact, we felt, in the beginning, that the development of a standard test was essential because, with our qualitative tests, we have not been in a position to freely accept applicants whose specimens have shown very faint traces of albumin. I am submitting to each of you a photograph of a table based on the work done in the laboratory of the Mutual Benefit Life Insurance Company in comparing the two commonly accepted qualitative tests and the test which we have developed. Assuming that we are justified in ignoring amounts of albumin which are less than .01%, we have had reported as positive with the nitric test, a total of 12 plus 7 plus 65 plus 55, or 139 cases, which, with the sulphosalicylic acid test would have been reported negative. On the other hand, there were reported negative with the nitric test, a total of 57 plus 6 plus 1 or 64 cases which would, with this test be reported as containing a small amount of albumin. At the bottom of the chart you will see similar figures for Purdy's test. 54 cases were reported negative to Purdy's test which were reported positive to sulphosalicylic acid. On the other hand, 221 were reported positive with Purdy's test which would be regarded as negative to the new test.

Lacking standards for our qualitative tests we encounter great difficulty in arriving at accurate estimations. The inherent fallacies long recognized, for example, an improper control of the acidity, also lead to erroneous results with the qualitative tests. For example, if you will kindly observe those specimens reported by the sulphosalicylic acid test as

containing .04% albumin, you will note that there were a total of 33 cases which, by the nitric acid test, were reported as follows: negative, 1; very faint trace, 1; trace, 6; moderate amount, 24; large amount, 1. You can see therefore that our qualitative tests are of questionable value as quantitative tests.

We wish to demonstrate an instrument which was developed by us with the hope that it might be of value in both qualitative and quantitative analysis, and after much difficulty, we have developed the instrument which is before you, based upon the principle that we must have a uniformly disseminated light, a daylight effect (secured by the use of an English daylight plate glass) and a uniformly black background. After experimentation with many types of electric light bulbs, we finally decided to use ordinary automobile 4 candle power lamps with automobile sockets, wired in series. Our experiments with a single long tube did not prove successful because the degree of illumination was not sufficient to enable us to use a daylight filter. Shorter tubes, giving a much greater light, were disappointing because of the great amount of heat developed.

Let me emphasize that this instrument is not an essential part of the equipment. A simple black test tube rack supplied with a black background is sufficient. In smoky cities and in poorly lighted laboratories, we believe the instrument will be of great value.

I take pleasure in turning over the demonstration of the precipitated urines to Dr. Kingsbury.

DEMONSTRATION AND REMARKS BY DR. F. B. KINGSBURY.

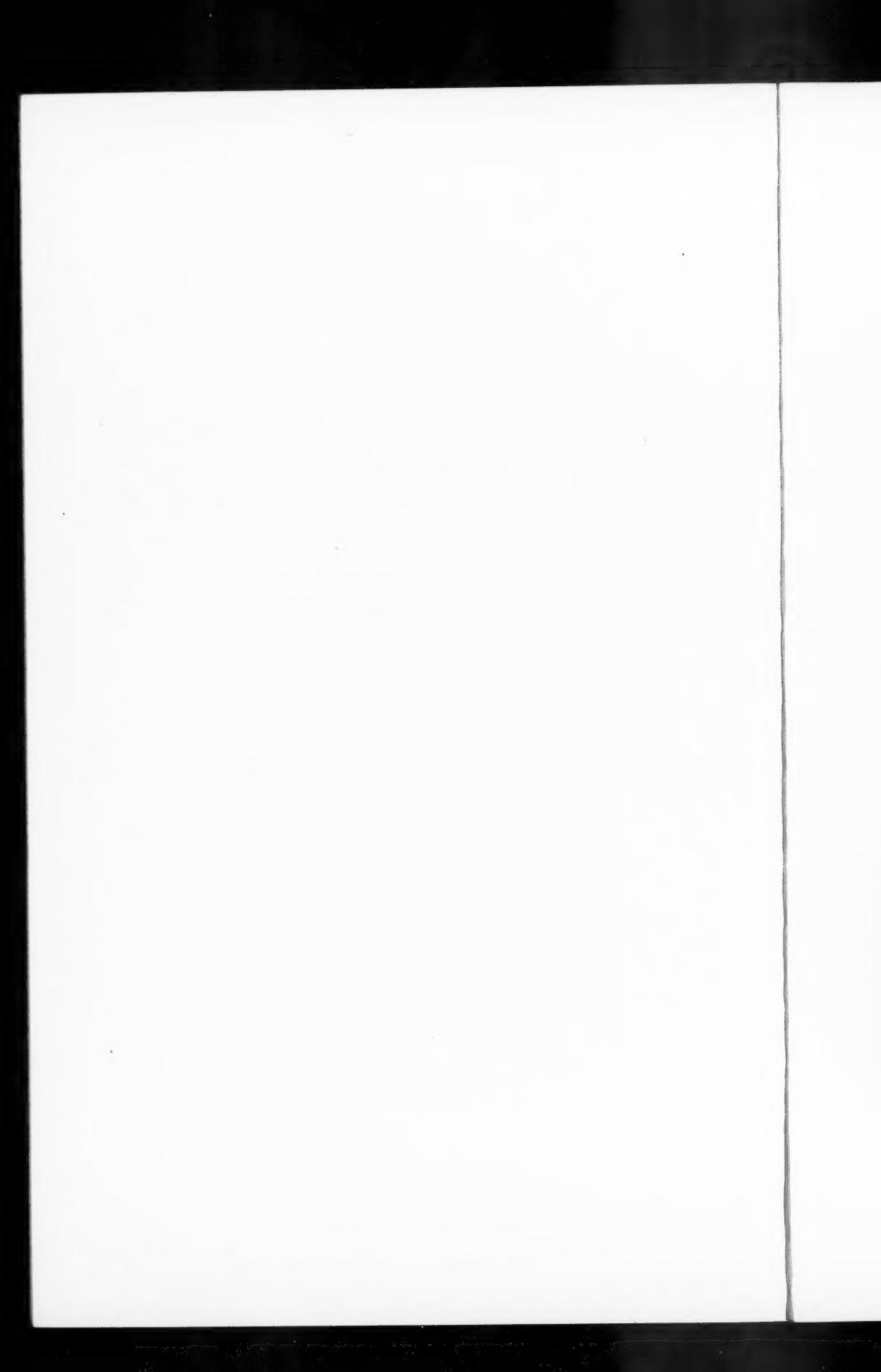
The standard tubes, as yet in the experimental stage were made by dispersing dialyzed suspensions of the sulfosalicylates of the serum proteins in 10% gelatin. The excess of sulfosalicylic acid used in making these compounds from diluted sheep serum must be removed by dialysis or by washing until

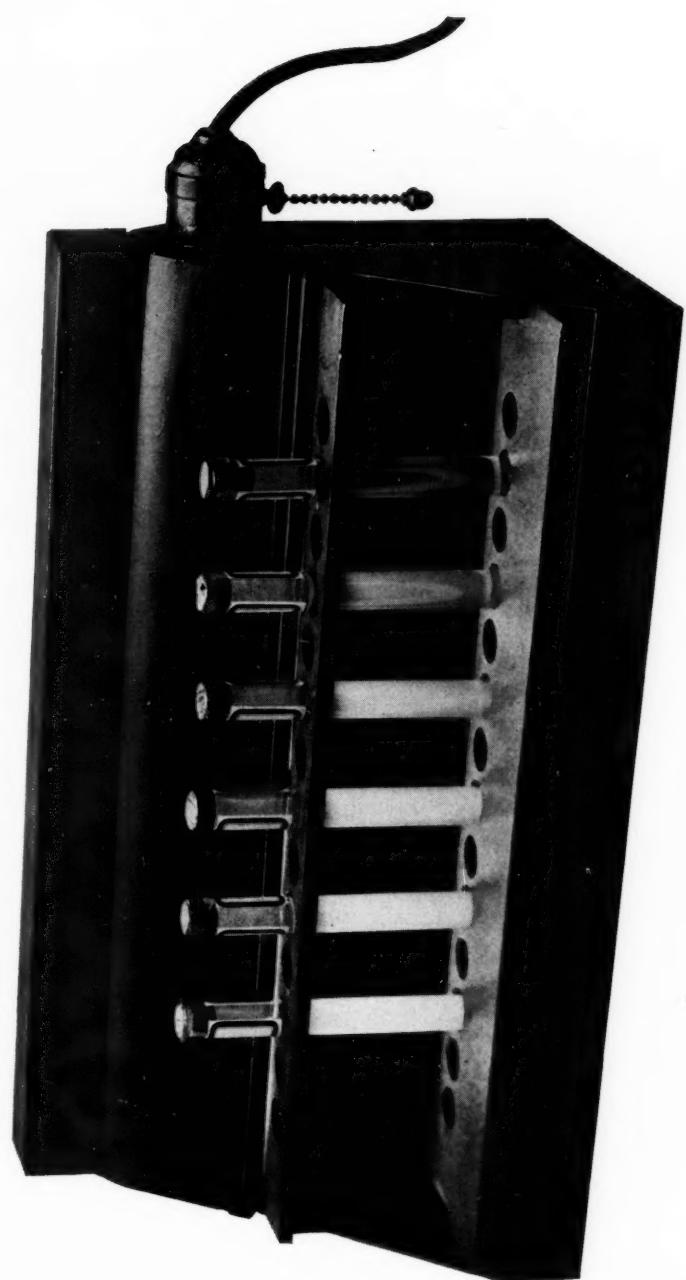
HELLER'S TEST.HEAT. SALT. ACETIC.

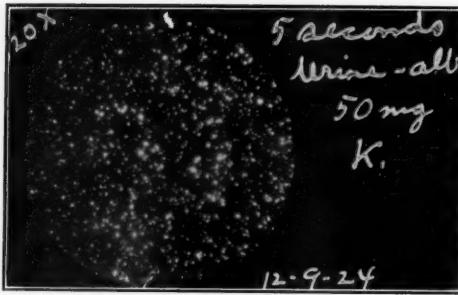
| <u>BY SULF</u> <u>AC/D</u> | <u>10</u> | <u>CASES</u> | <u>NEG</u> | <u>X</u> | <u>XX</u> | <u>XXX</u> | <u>XXXX</u> | <u>1/EG</u> | <u>X</u> | <u>XX</u> | <u>XXX</u> | <u>XXXX</u> |
|-------------------------------|-----------|--------------|------------|----------|-----------|------------|-------------|-------------|----------|-----------|------------|-------------|
| 0 to .003 | 3/35 | 3116 | 12 | 7 | | | | 3073 | *4 | 22 | 40 | |
| .004 to .008 | 547 | 427 | 65 | 55 | | | | 388 | 15 | 140 | 4 | |
| .009 and .01 | 158 | 57 | 38 | 54 | 9 | | *3 | 47 | 5 | 78 | 29 | 1 |
| .02 and .03 | 87 | 6 | 2 | 46 | 33 | | 6 | | 19 | 60 | 2 | |
| .04 | 33 | 1 | 1 | 6 | 24 | 1 | | | 4 | 22 | 6 | |
| .06 | 20 | | | 1 | 15 | 4 | | | 1 | 13 | 6 | |
| .08 | 15 | | | | 9 | 6 | | | | 8 | 7 | |
| .1 AND .15 | 16 | | | | 6 | 10 | | | 1 | 1 | 14 | |
| .2 | 10 | | | | 3 | 7 | | | | 10 | | |
| .3 | 2 | | | | 1 | 1 | | | 1 | 1 | | |
| .4 | | | | | | | | | | | | |
| .5 | | | | | | | | | | | | |
| .6 | | 1 | | | | 1 | | | | | 1 | |
| TOTAL | 4024 | 3607 | 118 | 169 | 100 | 30 | 3515 | 40 | 283 | 138 | 48 | |

Assuming that amounts of albumin less than .01% will be considered negative, we then have:
 *1 Reported negative to Heller's Test but positive to Quantitative Test 64
 *2 Reported positive to Heller's Test but negative to Quantitative Test 139

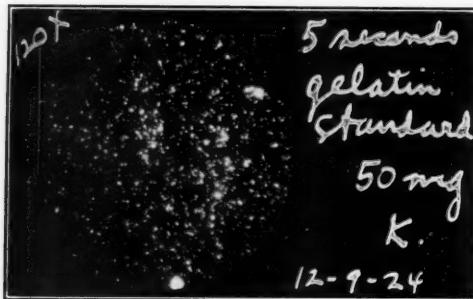
*3 Reported negative to Purdy's Test but positive to Quantitative Test 54
 *4 Reported Positive to Purdy's Test but negative to Quantitative Test 221







Urine containing .05% albumin precipitated in the same manner as demonstrated 120 X magnification, 5 seconds exposure.



Serum protein Sulfosalicylate in 10% gelatin. .05% standard tube. 120 X magnification 5 seconds exposure. Contains 2 large particles which are artifacts.

the hydrogen in concentration of the suspension is so reduced as to have little, or no effect on the gelatin, otherwise subsequent chemical action of the sulfosalicylic acid, which is a strong acid, on the gelatin medium may be expected. One drop of formaldehyde was used in each tube to preserve and harden the gelatin. The tubes are permanent for at least six weeks, which is as long as we have yet had opportunity to observe them.

It will be noted that the substances suspended in the gelatin are very similar to if not identical with those formed in the actual precipitation of the albuminous urine. Thus, the urine albumin tests look like the corresponding standards in daylight or in artificial light, whether examined by transmitted light (turbidimetrically) or by a reflected light (nephelometrically). When examined under the microscope (dark field illumination) at equal magnifications urine albumin tests and gelatin standards show a striking similarity in the size of the particles. Photo micrographs showing this are included in this article. While it is possible to use suspensions which have much smaller particles than those in the standard tubes exhibited, as was successfully done for many months in this laboratory, these standards, gum mastic in water, as well as certain others having particles of similar size to those of the mastic could be compared with urine albumin tests successfully only by reflected light. By transmitted light there was no resemblance whatever. It appears advantageous, therefore, to make standards which are as closely like the actual urine albumin tests as practicable. This had been accomplished by the writer. The permanency of these standards beyond six weeks remains to be demonstrated.

(Dr. Kingsbury demonstrated the estimation of albumin in seven specimens of urine.)

Dr. Ward—May I ask Dr. Cook to enlighten us somewhat as to this albumin question?

Dr. Cook—Mr. President—I think that any verbal expres-

sion of appreciation of this work would be grossly inadequate, because it seems to me there has never been a contribution to the Medical Directors' Association—and I believe also we should not lose sight of the fact that it is going to be a contribution to clinical medicine—which is of as great importance as this effort to obtain a proper numerical standard for the quantitative analysis of both albuminurias and glycosurias, so I will not even attempt to express my feeling in regard to its value.

As I understand it, Dr. Ward, this report of the Committee's is up for adoption?

Dr. Ward—Yes, I presume so.

Dr. Cook—I would wish very much, gentlemen, that we might not take these standards and attempt to operate with them during the coming year, until the Committee had gone a little further. Dr. Folin has pointed out of course that there are various uncertainties with regard to the stability of the reagents and the materials, and certainly the Committee has not felt justified in expressing themselves in regard to a definite use of any one of these three methods, and if they cannot, I do not see how any of us can. I would rather leave it to the judgment of this Committee to proceed until such time as they felt that they could bring before us a standard that they could recommend, and which had persisted for longer than three weeks, or some such period, so that we could take over in our own laboratories with the utmost confidence the test recommended by this Committee.

In regard to the recommendations of Dr. Patton's Committee on standards, I personally would regret excluding everything below .01%. Perhaps it does not make any material difference in my office—we might fail to catch a few cases of nephritis, and that would not matter in the ordinary run of cases, but if we are attempting to come to definite and comparable numerical standards, why lose track of the lower percentages? There is as yet no statistical evidence

that they do not prove to have a certain significance, particularly with regard to the older ages. For instance, if I were trying to select and to rate—and this is more important with the company doing substandard insurance where we are attempting to grade these cases numerically—a man sixty years of age, for example, applying for a large policy—with an occasional hyaline cast, a blood pressure slightly above or on the border line, and I do not know whether to take him at a rating of standard, or 125 or 150, and I know positively that the examination reported by the previous M. I. B. did not show albumin, it would be of very great help, and if they had found as small an amount at .01%, I would like to know that. I wish also it might be possible to subdivide that medium group. There is a tremendous range between .05 and 0.1, and I feel that we have made such a wonderful advance in this numerical determination, that it is a pity to lose sight of it by such a broad grouping as this.

Dr. Patton—The first division is from .01 to .05%, the second from .051 to 0.1% and the third is above 0.1 or over 100 milligrams.

Dr. Cook—I am just wondering if it would be possible to make a smaller division, in order that we might know more definitely what we are dealing with.

Dr. Patton—in regard to less than 10 milligrams, we are keeping a record, and we will have for our own use a standard that will record less than 10 milligrams. If there is any question at all of pulse, blood pressure, heart or low specific gravity, we are very likely to go back for a second specimen before we make a final decision.

Dr. Cook—Yes, but we would not get the benefit of it.

Dr. Knight—The M. I. B. is of course not a statistical report. It is a red flag.

Dr. Cook—Yes, but we would not get the red flag.

Dr. Ward—May I make this inquiry? Is it the judgment of the Committee that, of course, assuming that it will be a

256 Thirty-Fifth Annual Meeting

considerable length of time before all the companies are going to use the sulphosalicylic acid test, this discussion originally arose as to whether urine which did not contain sufficient albumin to show reduction by the nitric acid contact test should be reported? Now is it the advice of this Committee, Dr. Patton, that until this more refined method is adopted, that specimens examined in the laboratories of these various companies should not be reported, unless there is a sufficient amount of albumin in them to show by the contact test—in other words, that the heat test of itself should not be reported unless corroborated by the nitric acid test?

Dr. Patton—Yes, that is the suggestion of the Committee. We cannot require the companies to do anything—we can merely suggest, and we suggest that the companies do report along that line. I am frank to say from over thirty years' experience with the Heller's Nitric Acid Test, that I do not believe that a very faint trace as reported by any observer with the Heller's Nitric Acid Test is worth considering from a life insurance standpoint. If you have less than ten mgms. for one hundred c. c. urine, I would disregard it. I do say that in our own office, if we have low specific gravity, or blood pressure, we go back and get another specimen, but if we still find less than ten milligrams we disregard the albumin, even if on two specimens there is the very faintest trace. Now based on our 1917 report that was adopted as a standard by this Association, technically we should report as an M. I. B. report those amounts. Some of the companies have not been considering these from an insurance standpoint.

Dr. Rockwell—In 1917, when we adopted that report, I think there was no provision made for the quantitation to .01%. As I understand it, .01% represents the least possible thing that you can see with the Heller's Test.

Dr. Patton—We would not see it, Dr. Rockwell.

Dr. Bradshaw—if the Association adopts this method of

determining albumin, why not report the actual finding? What objection would there be?

Dr. Rogers—My reaction, when the suggestion was made, was that if we add all these refinements to the card, you will presently have a very cumbersome card, and as the object of the M. I. B. is simply to give a warning, it seems as if we go into elaborate details we are going a little too far. If any Company is interested in a case and wishes to know the details of the findings of another Company, it isn't a difficult thing to write and ascertain them. We are always glad to give details, and we take advantage of the courtesy of other Companies to ask them for details. I think the M. I. B. Committee has striven to keep the card as simple as possible. The fact that a high blood pressure has been reported in detail is of course an argument in favor of Dr. Bradshaw's suggestion. We could, however, load the card up with details regarding various impairments. My own reaction is that if we put out the red flag the details ought to take care of themselves. I think that covers the ground of Dr. Bradshaw's remarks.

Dr. McCrudden—As I understand it, the intensity of the precipitate depends upon four factors, the temperature, the amount of albumin present, the amount of acidity, and the presence of minute quantities of other salts, calcium, magnesia, potassium and sodium. The presence of those things in minute quantities influences a great deal. Now in determining the amount of albumin, all the factors are controlled, the temperature, the amount of acidity, the amount of albumin, except the amount of salts present in the urine. Now different urines contain different amounts of those salts, and also traces of possible colloid substances which might give variable results, even though the amount of albumin in the specimen in two different urines was the same? I think perhaps the difficulties which the Prudential found in their early test was a little indication of that? Is that so?

Dr. Folin—On the contrary, the variations in salt content have no effect. You can add as much as 5% of sodium salt or potassium salt and considerable calcium, and it has no effect on this reaction, but the color is an obscure problem, and that we cannot say, but so far as those other variants are concerned they do not come in, and therefore the values are reasonably uniform.

Dr. Benedict—Mr. Chairman—Dr. Cook has in a way put me up to backing him up on one point, although it is quite spontaneous on my part, and that is, in connection with the general view of one who has meddled more or less with methods, and not with regard to the albumin question at all, and that is the undesirability of recommending very definitely or putting out in definite form three different methods, the question still being open of a permanent standard for any one of the three, and as a matter of general principle I would urge the Association strongly to postpone the adoption of this report definitely, until the Committee has gone further in the matter and is able to recommend one method as probably the preferred one, and give some more definite information as to the permanence of the standards.

Dr. Folin—I think it is not very important whether you adopt this report or not. The substance has got into your heads. But quite aside from that, anything recommended is always understood to be tentative. One thing that does make a difference in the result is the presence of mineral acids, and that was really the cause of the difficulty that the Committee ran into, and when sulphuric acid was eliminated from the middle reagent, then the three different methods gave the same result, so that of these three methods, only one is comparatively new. One of them is a year and a half or two years old and the other one is several years old, so that there is not much uncertainty as to the results obtained by these processes. It is purely a question of the variant, the coloring matter in one of the reagents, otherwise they give, as

the Committee has shown, substantially the same result, but whether you adopt the report or not isn't very important, and I am perfectly willing personally to vote against the adoption, if that is the way the wind blows.

Dr. Ward—I do not know, gentlemen, that it is necessary to adopt the report. If we wanted to adopt any of it we could adopt that portion in which are defined the limits for the N, M or L.

Dr. Patton—I think all three members of the Committee as well as the laboratory workers who worked with the Committee feel that the Association is in position to adopt the N, M, or L limitations, and we feel that any one of the three methods if it were made the official Association method would give results so that any Company could act on those results without doing any harm to its mortality experience, but there is of course this feeling amongst any group of individuals—they get a liking for a certain method or a certain technique, and I do not believe that any one test adopted by this Association would be used universally by the member Companies. You know what happened with the Heller's Nitric Acid Test which came down from time immemorial. Soon after we adopted that, a number of us began growing away from it. We hope that with the sulphosalicylic acid that for all practical purposes we have the best precipitate for albumin for life insurance work. As Dr. Folin has well said, a slight variation in the percentage strength of the sulphosalicylic acid, or the addition of one or two other little things from time to time, is not going to make any material difference in the results.

Dr. Ward—It might be advisable to adopt a motion that where quantitative tests are employed, the N, M and L shall be as recommended by the Committee; where they are not employed that would not affect that Company at all.

Dr. Patton—Accepting the report does not mean that the

260 Thirty-Fifth Annual Meeting

Association adopts all the recommendations, because they were not made in the way of definite recommendations.

Dr. Rogers—Mr. President—I have been out of touch with the affairs of the Association for a time, and I rise to ask if my impression is not correct that this whole subject came up by reason of the circumstance that there was a feeling that a pretty large number of cases which were reported under our code symbols, on investigation were found not to have come within Heller's test at all, but had been reported as the result of the use of finer reagents. Now the Companies that used these finer reagents went right on and accepted these risks as if there were no albumin present, and yet they reported albumin, and so frightened, if you please, other members of the Association. That, as I understand it, was what raised this whole question. If that is so, and if this Association wishes to postpone action on the report of the Committee and wishes that Committee to study the matter still further and make a further report, would it not be well for the Association to adopt now a minimum beyond which albumin shall not be reported? I think that is the crux of the whole situation, and if that is so why should we not accept that to mean—"a clear reaction by Heller's Test"? If the Association adopts that now as its minimum, we shall clear the reports for the future of these delicate reagents that have been bothering us.

Dr. Ward—Your recommendation, Dr. Rogers, would be that we should pass a resolution that albumin should not be reported unless it can be demonstrated by the Heller's Test or its equivalent, and that any Company using a finer reagent than Heller's Test shall not report those minimum amounts of albumin which have heretofore been the cause of so much trouble.

Dr. Knight—I think the whole room would be in accord with adopting the second paragraph of the report, in which the Committee recognizes that some of the Companies are us-

ing the quantitative and some the qualitative, and is trying to get the same measurements, whichever method we use, and to mean the same thing by N, M and L. We are not changing the method at all, and therefore the Committee recommends limits for N, M and L records. It is the same thing. We are simply trying to speak a language that will be as reasonably uniform as we can make it, while we are using these different methods. I move therefore the adoption of the second paragraph of the Committee's report, as follows:

"We therefore recommend * * *

2. The following limits for N, M and L records:

| | | % | Mille % | Heller's Test |
|---|----------|-----------|----------|-------------------|
| N | Small | .01 - .05 | 10- 50 | Faint trace—trace |
| M | Moderate | .051-0.1 | 51-100 | Moderate |
| L | Large | Over 0.1 | Over 100 | Large |

Less than .01% or 10 mgm. per 100 c. c. urine should not be reported as an impairment."

Dr. Rogers seconded the motion, and it was carried.

Dr. Brown—Mr. President—I would like to move that the Committee on Urinary Impairments, whose report we have heard this morning, be continued, and that Dr. Ward, who has acted upon this Committee in an ex-officio capacity, be continued on that Committee.

Dr. Patton seconded the motion.

Dr. Cook—I would like to suggest as an amendment to that motion that the Secretary be instructed to spread on the minutes of the meeting an expression of the appreciation of this Association of the work of the Committee.

Dr. Rogers seconded the amendment which was adopted and the motion, as amended, was carried.

AFTERNOON SESSION

Dr. Ward—Gentlemen—The first paper we are going to take up this afternoon is entitled—"Some Non-medical Thoughts Regarding Selection." I am sure you are all acquainted with the gentleman who is to present this paper to you, because he has been in close contact with the medical work of the Companies for many years. In 1909 Mr. Rhodes was a member of the Actuarial Committee which, with the Medical Directors' Committee, formed the Medico-Actuarial Committee. I feel therefore that I am not introducing a stranger to you in presenting Vice President Rhodes, of our Company.

Mr. Rhodes—Mr. President and Gentlemen—it gave me great pleasure to accept Dr. Ward's invitation to speak this afternoon, because it afforded me an opportunity that I have coveted to acknowledge in person my very high appreciation of the honor which you conferred upon me last year, in electing me one of your Honorary Members.

SOME NON-MEDICAL THOUGHTS REGARDING SELECTION.

MR. E. E. RHODES.

With our present ideas regarding selection, what *a priori* conclusions should be reached concerning the mortality under the early policies issued by one of the oldest companies in the country when we know that the application blank did not contain much of the data now deemed essential, that both the officers and agents were without any life insurance experience, and that the rate of rejection was exceedingly low?

The policies issued by the Mutual Benefit in the years 1845-1856, inclusive, have all been terminated. We have, therefore, for these policies a complete mortality experience, embracing 8,560 policies and \$26,716,025 of insurance, and

covering seventy-three policy years. So far as I know, no such experience has been published, and the results may therefore be of interest to the Association of Life Insurance Medical Directors.

By adding the exposures for the several years we find that for the entire experience the number of policies at risk was 125,489, and the amount of risk was \$391,522,629. 3,106 policies, insuring \$9,633,152, ceased by death. The percentage of actual to expected deaths by the American Experience Table was 93 by policies and 94 by amount. During the first five policy years these percentages were 91 and 85, respectively. By the American Men Ultimate Table the percentages for the entire experience were 106 by policies and 108 by amount.

It will be interesting to note the ratio of actual to expected deaths according to several age groups by both the American and American Men Ultimate Tables. This is shown by the following summary:

| Attained Ages | American % | AM % |
|------------------|---------------|---------|
| 20-29 | 147 | 277 |
| 30-39 | 106 | 196 |
| 40-49 | 94 | 136 |
| 50-59 | 93 | 101 |
| 60-69 | 76 | 75 |

It will be observed that at the younger ages the actual deaths very much more closely approximate the expected deaths by the American Table, while at the older ages they are substantially the same by both Tables. This is due to the fact that the rates of mortality by the American Men Table, expressed as percentages of those by the American Table, are as follows:

| Ages | % |
|-------|-----|
| 20-29 | 53 |
| 30-39 | 54 |
| 40-49 | 69 |
| 50-59 | 92 |
| 60-69 | 101 |

264 Thirty-Fifth Annual Meeting

In 1857 the company's mortality experience during the first eleven years was tabulated according to the Carlisle Table of Mortality, upon which the premiums were based. The actual deaths were 88% of the expected. According to the American Experience Table the actual deaths for the first eleven years were 102% of the expected, and according to the American Men Ultimate Table they were 144%.

On the basis of the Carlisle Table the Company showed a mortality profit for the first eleven years. If the American Table had been in use the Company would have come out about even, but if the American Men Table had been in use, the Company might have been forced into insolvency.

In the earliest application form used by the Company there was no medical examination. The applicant was required to state whether he had had smallpox, gout, rupture, fits, dropsy, asthma, liver complaint, consumption, spitting of blood, disease of the heart or any of the vital organs, or any severe or constitutional disease, and whether he had ever met with any accidental or serious personal injury. The applicant's physician was called upon to answer similar questions and also to state whether the applicant was sober and temperate, whether there was any hereditary predisposition to disease, whether the applicant was accustomed to much exercise and whether the physician was aware of any particular circumstances tending to shorten the applicant's life. No statement as to height and weight was required.

Beginning in 1846 the company obtained reports from medical examiners, but they were very meagre. The examiner was only asked to report on the applicant's height, figure, general appearance, whether there were any signs of affection of the head or of predisposition to it, the stethoscopic character of respiration, and heart's action, the rate and character of the pulse, whether there was an habitual cough or expectoration, occasional difficulty of breathing, or palpitation, and any signs of abdominal or other disease.

The weight was not required until 1858 and it was not until 1865 that the family history was required. An examination of the urine was first required in 1879 and then only when the amount of insurance reached \$10,000.

During the first twelve months that the Company was in business it received 1,723 applications and declined 9, or 1/2 of 1%. During the years 1845-1856, inclusive, the Company rejected 235 applications, or 2.06% of the number submitted. The average rate of rejection for standard insurance at the present time is about 9%.

It will seem surprising that under the conditions the mortality was fairly favorable. Bearing in mind that the agency force was untrained, we may conclude that there was little, if any, preliminary selection on their part. The home office had a very incomplete picture of the applicant. The standards of selection, if there were any at all were very liberal as shown by the low percentage of declined cases. I have examined several of these declined applications and I cannot see wherein some of them were not as acceptable as those which were accepted.

I think I have shown that the principles of present day selection were not essential to the safe conduct of a life insurance company in the middle of the last century. Lest, however, I be misunderstood, let me hasten to say that I regard the medical boards of life insurance companies as a very necessary part of the organization today. Safety and prosperity are not synonymous, although they went hand in hand in the early days of the Mutual Benefit. There can be safety without prosperity, but there cannot be prosperity without safety. The function of the medical board is to link the two. The lowest death rate is not evidence of the soundest underwriting.

In the early days of the business commissions and other expenses were very low. Policies were contestable at any time, and by their terms were forfeited upon nonpayment of any premium. Premium rates were high and comparatively little

stress was laid upon dividends. The profits of that time have disappeared and the situation today calls for the highest degree of intelligence in the selection of risks. Upon the standards of selection adopted by a company depend, not only the safety of the company, but its growth and prosperity as well. These standards are inextricably bound up with the different plans of insurance, the reserve basis, the commissions paid agents, and the dividends allowed to policyholders. The higher the commissions the lower will be the dividends unless the difference between a high and a low commission scale is made up out of additional mortality savings. This means in turn the exclusion of all but gilt edged risks, and this is followed by a narrowed field of activity for the agency force, unless a company does a substandard business. What I have said above with regard to companies doing business on the participating plan applies equally to those doing business on the non-participating plan. High expenses and liberal standards of selection mean high premium rates if there is to be a profit for the stockholders.

The plan of insurance favored by a company has a very material bearing upon the mortality savings. Let me take ordinary life, twenty-premium life, and twenty-year endowment policies, all issued at age 35, for \$10,000. We will assume that the company operates under the full net level premium reserve on the basis of the American table and 3% interest. The sum of the tabular costs of insurance for the first twenty years on the reserve basis mentioned would be \$1,930.63, \$1,591.29 and \$1,122.53, respectively, for the three plans. If the same death rate should be experienced under the three plans, the ratio of the actual to expected cost of insurance for a period of years would be different under the different plans. For example, if the death rate during the twenty-year period was in accordance with the American Men Select Table, the percentages of the actual to expected cost of insurance would be 70%, 68%, and 63%, respectively. On

this basis the mortality savings under the three plans would be \$573.75, \$509.35, and \$420.39, respectively. The reason for these differences is that because of the larger reserves on the higher premium policies, there is under them a smaller amount at risk, and consequently a lower tabular cost of insurance. To have the same savings under the twenty-premium life and twenty-year endowment that are shown for the ordinary life, namely, \$573.75, the mortality under the twenty-premium life policies would have to be 94%, and under the twenty-year endowment 78%, of the American Men Select Table.

The American-Canadian Mortality Investigation showed a lower mortality on twenty-premium life and twenty-year endowment policies than on ordinary life policies. The difference was not very material and no larger than might be expected when the higher premium plan was desired by applicants who regarded themselves as particularly good risks. It might not be realized by a company if the agents, because of the difference in commissions, influence the writing of the higher premium policies.

We may conclude that companies desirous of making a favorable showing with regard to dividends, or of realizing handsome returns for stockholders, should endeavor to have a preponderance of ordinary life policies or of long term endowments, or should adopt stricter standards of selection if they favor the high premium plans. It would seem to follow logically that term policies would be the most profitable for a company. This would be true if the matter of adverse selection could be eliminated, and if it were not for the fact that any additional savings in mortality would be more than offset by the expense of procuring enough new insurance each year to maintain the premium income.

It is a simple matter for a company to throw the weight of business wherever it desires. Some years ago the Mutual Benefit decided that it was not getting a sufficient proportion

of its business on the ordinary life plan. At that time 29% of its new issues were on the whole life plan, 55% on the limited payment life plan, 10% on the endowment plan and 6% on the convertible non-renewable five-year term plan. The desired change was brought about by a comparatively trivial adjustment of the commission scale. The result is that in 1923, 72% of the new issues were on the ordinary life plan 19% on the limited payment life plan, 5% on the endowment plan and 4% on the five-year term plan. It is interesting to observe that while in numbers 72% of the new issues of 1923 were on the ordinary life plan, 80% of the new insurance was on that plan, and that while in numbers 19% of the new issues were on the limited payment life plan, only 12% of the amount of new insurance was on that plan. The amount of new insurance issued under the endowment plan was 3% of the total. This change has increased very considerably the company's surplus earnings from mortality, irrespective of what the general death rate might be. It has also advantaged the company otherwise in matters apart from that of selection of risks.

A company which uses a lower reserve basis than another company may be more liberal in its selection, and have the same surplus earnings from mortality, other things being equal. This is for the reason that I have set forth above in discussing the difference between various plans of insurance. The low reserve company may therefore experience a higher death rate and still have the same mortality earnings as a high reserve company with a lower death rate. Much stress, however, cannot be laid upon a difference in the reserve basis.

A company which is growing very rapidly and which maintains the full net level premium reserve, cannot afford to be as liberal in its selection as one which is experiencing a normal growth. This is for the reason that an undue amount of new business requires a considerable investment of funds, and sat-

isfactory returns upon this investment can be realized only through a low death rate coupled with a low lapse rate.

Speaking generally, it is practicable for a company to set the death rate at any figure which it desires to realize and to accomplish within reasonable limits the desired result. When the standards of selection are such that it is able to furnish insurance at a reasonable cost to all who are entitled thereto, it has done all that can be fairly expected of it. Having adopted a certain standard it will be necessary to accept some risks upon which the mortality will be higher than the standard. Otherwise the death rate will be lower than the desired standard for the reason that the company will insure some risks upon which the death rate will be lower than the standard, and the standard cannot be maintained without the inclusion of the poorer risks. Skill in selection is shown by the coolness of the actual results to the desired standard.

Through the substitution of facts for impressions, the work of the Medical Directors' Association has acquired a very scientific aspect. The day when a stout medical director thought that stout men were the only good risks and when a lean director thought that lean men were the only good risks has passed.

The days of progress are not over. Although we know fairly well the effect upon the death rate of a single impairment we have very little knowledge of the effect of a combination of two or more impairments. Perhaps Dr. Rogers and Mr. Hunter will divulge more of this information in the near future. They are the hope and the despair of every student of selection. We hope that for many years to come we shall receive in ever growing measure the results of their researches, but we despair of ever knowing as much of the subject as they know. You will understand why I have skirted around the fringe of the subject and confined myself to certain generalities.

270 Thirty-Fifth Annual Meeting

Dr. Ward—I know that you will all take this paper home with you and ponder over what Mr. Rhodes has said, and get a great deal of good from it. We now come to a very interesting subject and that is the very complex problem of goitre. This subject has been brought to the attention of the Convention before, and we hope to get some further light upon it this afternoon. Dr. Carber will present his paper to us:

THE PRESENT STATUS OF GOITER.

DR. FRANK H. CARBER,

Medical Inspector, The Mutual Life Insurance Company.

Medical directors are not willing to assume for their companies, the hazard involved in the acceptance of an applicant with a goiter, who shows evidence or gives recent history of thyroid dysfunction or local pressure.

The problems of selection are not solved, however, by the exclusion of this group: Indiscriminate acceptance of all non-toxic goiters would inevitably result in alarming increase in the mortality of the class, since many non-toxic goiters are potentially very dangerous. The danger can be eliminated only by careful selection, utilizing judgment which is based upon thorough pathologic and clinical knowledge, and information which is at least, reasonably accurate.

It is the object of this paper to submit a classification of all goiters which is based solely on their histopathology, to outline the physical and clinical characteristics of each type so classified, and to evolve a questionnaire for the examiner or attending physician which will bring out the physical characteristics and clinical history of each case, clearly enough for use as a basis for intelligent classification, and final disposition by the medical approving officer.

This classification is based upon three histologic factors as

outlined by Plummer (1) of the Mayo Clinic after clinical and microscopic study of several thousand cases in which the gland was removed. It has been recently employed by Coller (2) of Ann Arbor in a discussion of the morbidity of a large group of endemic goiters in Michigan.

The histologic factors which form the basis for this classification are:

1. The number of alveoli.
2. The character and amount of alveolar epithelium.
3. The amount of intra-alveolar colloid.

The term *Hypertrophy* is here used to denote increase in size of the gland caused by increase of the non-functioning elements, principally the colloid.

The term *Hyperplasia* denotes increase in the functioning elements, namely in the number of alveoli and in the number and size of the epithelial cells forming the alveolar lining. There is not necessarily an increase in the size of the gland.

TYPES OF GOITER

1. HYPERTROPHIC:

The predominating feature is increased intra-alveolar colloid. There is also perhaps, some increase in the number of alveoli. Hypertrophic goiter is the common adolescent goiter or so-called physiologic enlargement in early life. In later life, it is known as the simple colloid goiter. It is non-toxic, but frequently develops adenomata in the gland and is then designated colloid adenoma.

2. HYPERPLASTIC: (10% of all goiters.)

This type is characterized by marked increase in the number of alveoli and in the number of alveolar cells. The epithelium becomes high columnar in type, and because of cell proliferation the alveolar lining membrane becomes

272 Thirty-Fifth Annual Meeting

folded upon itself in such a way that it resembles, on section, an epithelial tumor. Hyperplastic goiter is clinically exophthalmic goiter. It is always toxic, with exophthalmos and nervous symptoms in predominance.

3. ADENOMATOUS:

Characterized by grouping of the alveoli with or without encapsulation: It is one of the commonest types of goiter. About 20% become toxic, many through hypersecretion of the alveolar tumors, according to Plummer (4), some because of hyperplastic change which may develop. There are three types of adenomata:

A. Simple Adenoma: (85% of all adenomata.)

This type has been histologically described above. Many of them become toxic because of hypersecretion of the alveolar tumors. The predominating symptoms are cardiac. There is no exophthalmos. The syndrome is called hyperthyroidism or thyrotoxicosis and is *not* the syndrome which occurs in exophthalmic goiter. Simple adenomata may become hyperplastic as described below.

B. Hyperplastic Adenoma: (5% of all adenomata.)

Histologically there is hyperplastic change in the parenchyma of the gland around the adenoma as described under 2. The syndrome of this type approaches that of exophthalmic goiter in direct ratio to the amount of hyperplasia in the gland.

C. Fetal Adenoma: (10% of all adenomata.)

Exists at birth. It is apparently a congenital "rest." Histologically composed of strands of embryonic cells. Regarded by some as more likely to become hyperplastic or to undergo malignant degeneration than simple adenoma.

4. CARCINOMA: (1.25% of all goiters.)

Very hard, nodular, rapidly growing tumor. Usually in later life. May begin as primary carcinoma or may be due to malignant degeneration of an adenomatous gland.

5. SARCOMA: (0.5% of all goiters.)

Very large, soft, rapidly growing primary tumor.

6. INFLAMMATORY: (1.25% of all goiters.)

Thyroiditis; acute or sub-acute with the usual phenomena of inflammation.

It will be noted that over 85% of all goiters are either hypertrophic goiters or adenomatous goiters. The relative proportions vary with the age. In the younger ages, about 65% are hypertrophic and 20% adenomatous. After age 40 the relative incidence is reversed, 65% are adenomatous and 20% are hypertrophic. The total goiter incidence in women remains the same from age 15 to 60. In men there is a gradual drop from age 20 to 40. The level after 40 in both instances is maintained by adenomatous goiters with the incidence of hypertrophic goiters at a minimum (3).

Each type under the above classification shows a predominance of change in one of the histologic factors mentioned by Plummer. It should be borne in mind, however, that there may be changes in two or even three of these factors in the same gland with resulting mixed types. Examples are the colloid adenomata which are simple colloid goiters containing adenomata, and hyperplastic adenomata, which are adenomatous glands in which there is hyperplastic change in the parenchyma.

Degenerative changes, cystic, malignant, fibroid, hyaline, hemorrhagic, or calcareous, may take place in all goiters but are particularly common in adenomata. They do not produce symptoms of intoxication but they may cause local pressure symptoms.

274 Thirty-Fifth Annual Meeting

Hypothyroidism is very rarely found in association with goiter. Plummer (4) states that he has never seen a goiter case with sufficient hypothyroidism to produce myxedema.

PHYSICAL AND CLINICAL CHARACTERISTICS

1. HYPERTROPHIC GOITER: Simple Colloid Goiter.

The gland is soft, smooth and symmetrical as a general rule. Occasionally the alveoli become so distended with colloid that some of them rupture and fuse to form a soft nodule.

Hypertrophic goiters are *never* toxic, but many of them develop adenomata, usually after age 25, which are *potentially* toxic. It is well known that adolescent goiters frequently become adenomatous, and that they have a tendency to liberate their colloid at about age 25 causing adenomata which hitherto have been unobserved to become apparent. The fact that in the younger ages 65% of all goiters are hypertrophic, while after age 40, 65% of all goiters are adenomatous, would lead one to infer that most of the hypertrophic goiters beyond age 25 ultimately become adenomatous.

The so-called physiologic enlargement or adolescent goiter is the most common type of hypertrophic goiter. It appears about the middle of the second decade. Most of them gradually liberate their colloid and regress before age 25. Those in which the alveoli have been markedly distended with colloid usually persist indefinitely as simple colloid goiters. Many develop adenomata which become apparent when regression takes place, and the goiter persists as an adenomatous goiter.

2. HYPERPLASTIC GOITER. Exophthalmic Goiter. Graves' Disease.

The gland is symmetrical, only moderately enlarged and is

very firm, almost to the point of hardness. The surface is smooth, and thrills and bruits are often demonstrated.

The average age at which the goiter is first noticed is 36, and symptoms make their appearance within a year. They run a course marked by acute periods of exacerbation of three to six months duration, called crises, which are followed by periods of remission of three months or more duration. In general the first few crises are most severe, and they gradually become milder, and the periods of remission longer.

The predominant symptoms are *nervous*, and should *not* be confused with hyperthyroidism from toxic adenoma, in which the predominant symptoms are *cardiac*.

We find cerebral irritability, tremors, flushing, sweating, exophthalmos and marked increase in the basal metabolic rate with loss of weight. The tachycardia is of nervous origin. Permanent cardiac damage occurs only in prolonged cases after several crises, or in those where the acute intoxication is overwhelming and weakens the heart muscle.

Thyroidectomy during the crises is disastrous, but the results of operation during a period of remission are excellent because there is no cardiac damage. The principal danger following operation is that of recurrence, which practically always takes place within five years, usually within the first two or three years.

3. ADENOMATOUS GOITER:

The adenomatous gland is rarely symmetrical. One lobe (frequently the right) or isthmus, is larger. It is generally nodular; the nodules may be single or multiple, localized to one lobe or diffused throughout the whole gland, and may vary in size from a pinhead to a hen's egg.

Adenomata of the thyroid are tumors, and should be so regarded. Histologically they are the same as adenomata of the breast or any other gland, but they do not have the same

276 Thirty-Fifth Annual Meeting

potential dangers. Although malignancy does occur, the principal danger lies in over activity of the alveoli or from hyperplastic change.

About 20% to 30% of all adenomatous glands become toxic, all except about 5% of the toxic cases being due to hypersecretion of the alveoli of the adenomata of simple type (4).

The average age at which adenomata are first apparent is 22. According to Plummer (1) they remain quiescent between 15 and 20 years. Many originate in simple colloid or adolescent goiters, but many are first noticed as localized swellings. At age 40 to 45 symptoms of hyperthyroidism or thyrotoxicosis appear. This syndrome should *never* be confused with Graves' disease. There is *never* exophthalmos, and the predominant symptoms are cardiac, tachycardias, arrhythmias, hypertrophy, and finally fibrillation and decompensation. The cardiac damage is permanent, and unoperated cases become chronic cardiopaths or die a cardiac death. Nervous symptoms are present in varying degree, the basal metabolic rate is increased, and there is loss of weight.

This is the worst class of goiters because of the cardiac damage, and they show the highest operative mortality and the lowest percentage of cures.

About 5% of all adenomatous goiters are associated with *hyperplastic* changes in the parenchyma around the adenoma. These cases present symptoms of exophthalmic goiter, including the exophthalmos, in direct ratio to the amount of hyperplasia in the gland. Occasionally an extreme case is seen, with a nodular goiter of many years duration who suddenly develops the entire syndrome of Graves' disease. If we think of the term *hyperplasia* as *always* being associated with symptoms of *exophthalmic goiter*, it is easy to understand such a picture.

Fetal adenomata probably are congenital. It is thought by some that this group is more likely to become toxic or to

undergo malignant change. They are often single, and sometimes are demonstrable in the first decade.

There are apparently other dangers than those of toxic origin, which exist in the latter part of the clinical course of adenomatous goiters. Coller (2) found enlarged hearts and complaints of palpitation and dyspnoea in about 50% of a series of nontoxic cases with normal basal metabolic rate, who were over 40.

It should be remembered also, that adenomatous goiters are more likely to undergo cystic, hemorrhagic, and malignant degeneration than any other type, and to produce local pressure symptoms.

CONCLUSIONS

From the foregoing, it would appear that the *period* of greatest hazard is after age 40 or 45, and that the nodular asymmetrical goiters present the greatest potentialities of danger, of all the nontoxic goiters.

It would also appear that the most favorable age is under 25, and the most favorable type of nontoxic goiters are those which are smooth and soft and symmetrical.

Most of the smooth symmetrical goiters in young persons will regress at age 25, and of those which do not, less than 20% will become toxic after they have been policyholders for 20 years or more. It would therefore appear that ordinary life contracts at standard rates might safely be offered to persons under 25 who have smooth, soft, symmetrical goiters.

It would also appear that contracts which terminate at age 40 or 45 might be offered with reasonable safety to persons under 25 who have nodular goiters which are not symmetrical, and to persons over 25 who have smooth, symmetrical goiters.

It would appear unsafe to offer any contract at standard rates to persons over 25 with asymmetrical nodular goiters,

or to any person having a goiter of any type who is over 40 years old.

Large goiters or cystic goiters cannot be regarded as standard risks because of the danger of pressure and intrathoracic growth.

Goiters which have not remained at their present size at least three years are not standard risks because of the danger of increased activity of the gland or malignant change. Exophthalmic goiters, cancer, sarcoma, and rapid cystic, hemorrhagic or malignant degeneration are thus guarded against. All hard goiters should be excluded.

No goiter cases presenting evidence or giving recent history of thyroid dysfunction or local pressure, are acceptable.

Too much stress cannot be laid upon the importance of extreme care in the cardiovascular examination of applicants with goiter who appear to be acceptable. Special attention must be given to the size of the heart and to the rate and rhythm of the pulse before and two minutes after exercise. Blood pressure readings should be required, and careful examination of the nervous system with special reference to tremors. In doubtful cases, where it is practicable, an estimation of the basal metabolic rate might be employed as a reserve precautionary measure.

THYROIDECTOMY

Cases who have had thyroidectomy for toxic adenoma or exophthalmic goiter, who can pass a satisfactory physical examination with the special precautions mentioned above, should be acceptable as standard risks five years after complete recovery. The principal danger, that of recurrence, has been eliminated after such a period of postponement.

Cases who have had thyroidectomy for nontoxic goiter who pass a satisfactory examination with above-stated precautions, give very little danger of untoward effects or complications two years after recovery.

All cases who have had thyroidectomy should show no signs of enlargement of the thyroid. The exophthalmos does not completely subside in most of the cases which are otherwise cured, and should not be regarded as a cause for rejection if there are no other physical abnormalities. No cases with evidence of hypersecretion are acceptable.

Hypothyroidism should be borne in mind in the selection of all cases who have had operation. However, it is a rather infrequent complication of modern goiter surgery. Careful cardiovascular examination, blood pressure readings and microscopic urinary examination will protect against the late degenerative effects of this condition, and the basal metabolism test would also be of great help in doubtful or suspicious cases.

The following questionnaires for examiner or attending physician are based upon physical characteristics and clinical history, as a practical method of obtaining reasonably accurate information.

QUESTIONNAIRE

Requirements for Goiter (No operation).

1. When was goiter first noticed?
2. How long present size?
3. Measurement of neck at greatest circumference?
4. Is gland firm or soft?
5. Is surface smooth or nodular? Size of nodules?
6. Is there any localized enlargement in either lobe or isthmus?
7. Is goiter substernal?
8. Any symptoms of local pressure on structures of the neck?
9. Any general symptoms of thyroid intoxication?
 - a. Exophthalmos
 - b. Loss of weight
 - c. Tremor
 - d. Palpitation of heart

- e. Sweating
- f. Nervousness

10. Re-examine heart.
 - a. Enlargement
 - b. Murmurs
 - c. Arrhythmia
11. Take pulse and blood pressure reading, before and two minutes after exercise.
12. Microscopic of urine.
13. Basal metabolic rate (in doubtful cases where practicable).

THYROIDECTOMY.

- I. Statement by operating surgeon:
 - A. Date of operation
 - a. Extent of operation
 - B. Type of goiter
 - C. Indication for operation
 - 1. Toxic symptoms? Describe
 - 2. Local pressure symptoms? Describe
 - 3. Cosmetic reasons? Describe
 - 4. Prophylactic reasons? Describe
 - D. Date of complete recovery
- II. Present condition—To be noted by Examiner:
 - A. Does any enlargement of gland persist?
 - B. Any evidence of intoxication now?
 - 1. Cardiac?
 - 2. Nervous?
 - 3. Is weight back to normal?
 - 4. Exophthalmos?
 - C. Any evidence of hypothyroidism?
 - D. Re-examine heart:
 - 1. Hypertrophy
 - 2. Arrhythmia
 - 3. Murmurs

- E. Blood pressure reading and pulse rate before and two minutes after exercise?
- F. Microscopic of urine?
- G. Basal metabolic rate in doubtful cases where practicable?

References:

1. Plummer, H. S. The Clinical and Pathologic Relationship of Hyperplastic and Non-hyperplastic Goiter. *Journal A. M. A.*, Aug. 30, 1913.
2. Coller, F. A. The Morbidity of Endemic Goiter. *Journal A. M. A.*, May 31, 1924.
3. Levin, Simon. One Thousand, One Hundred Forty-six Goiters in One Thousand Seven Hundred Eighty-three Persons. *Archives International Medicine*, Apr. 15, 1921.
4. Plummer, H. S. Inter-relationship of Function of the Thyroid Gland and its active agent, etc. *Journal A. M. A.*, July 23, 1921.

Dr. Ward—I congratulate Dr. Carber upon his remarkably clear differentiation. We cannot pretend to remember all those figures, but we have them in print, and as we have opportunity to study this paper, I am sure it will prove to be a subject that will give us a great deal to think about. I will ask Dr. Hutchinson to open the discussion of this most excellent paper.

Dr. W. G. Hutchinson—Our knowledge of goitre has been greatly enhanced in recent years. Without doubt the most important contribution to our knowledge was the discovery that an iodine shortage is the causative factor in the so-called simple goitres and the further fact that supplying the system with a proper amount of iodine at certain periods of life will effectively prevent the development of such goitres. This knowledge has aroused Public Health Authorities in the various endemic goitre areas to the importance of determining the extent of the disease and instituting measures for its prevention. A number of surveys have been made in various districts and the results published, which are undoubtedly familiar to you all.

In our state of Michigan goitre is endemic. Under the

282 Thirty-Fifth Annual Meeting

direction of Dr. Olin, Commissioner of Health, a goitre survey was made in four counties of the state and the results published by Dr. Olin in the journal of the American Medical Association of April 26th, 1924. The counties selected were chosen after the determination of the iodine content of a large number of samples of ground water taken from different parts of the state. The survey was made among school children and the ages of those found with goitre varied from 5 to 18 years of age. The total number examined was 31,612 of whom 47.2% showed goitre. In this group there were 15,809 boys and 15,803 girls. Goitre was found in 40.5% of the boys and 53.8% of the girls. In the age distribution there was a steady increase in the number of cases up to age 10 in the boys and to age 13 in the girls, after which ages there was a steady decline in the curve. The number of cases found in each county was in inverse ratio to the amount of iodine found in the water of the county. The water of Macomb County contained 8.7 parts per billion of iodine and the number of children with goitre was 26%. Midland County water 7.3 parts per billion iodine, goitre cases 32.7%. Wexford County 0.5 parts per billion iodine, goitre cases 55.6%, and Houghton County, no iodine, goitre cases 64.6%.

During the winter of 1922-23 a goitre survey was made by the Medical School Inspectors of the Detroit Board of Health among the students taking gymnasium work in the Intermediate and High Schools of Detroit. This survey however was limited to the children who were sent to the inspectors by the teachers as suspicious of goitre. There were 8,815 boys examined of whom 725 or 8% had goitre, and 9,520 girls examined of whom 2,209 or 23% had goitre. Had this survey been made by the Medical Inspectors among all the students probably a larger number of goitres would have been found. In neither of these surveys were any cases of exophthalmic goitre found. These figures indicate somewhat the present status of goitre in the State of Michigan. Similar surveys

in other endemic goitre districts show fully as many if not more cases.

Goitres are primarily divisible into two classes the simple and exophthalmic. Exophthalmic goitre is a distinct clinical entity, primarily a constitutional disease in which the thyroid hyperactivity is of a secondary nature. It is always a toxic goitre, the toxicity however varying in different cases, and at different periods in the same case. The disease is progressive, and degenerative. Changes in the cardio-vascular system and other vital organs are always an end result.

The simple goitres may be divided for practical purposes into two classes (1) Colloid and (2) Adenomatous. Both of these types most frequently appear during the period of adolescence and the true colloid goitres usually automatically disappear before the age of twenty-five, without having caused any symptoms. The adenomatous type however do not disappear and either remain stationary or increase in size, not infrequently causing hyperthyroidism.

From an underwriting standpoint hyperthyroidism is the most important element of goitre cases. The degenerative myocardial and vascular changes which occur in this condition are too well established to require discussion. When such changes have occurred the damage is permanent and necessarily influence the longevity of the risk. Knowing that true colloid goitres rarely cause hyperthyroidism and that hyperthyroidism always accompanies exophthalmic goitre the problem of selection is largely limited to the intervening class of cases.

It is a fact that every goitre is a potential case of hyperthyroidism. This fact perhaps does not apply to cases of the true colloid type, but so many cases at first thought to be colloid, later prove adenomatous and possibly toxic that it practically applies to all goitres. It has been demonstrated that the most frequent type of goitre in the endemic goitre areas is a combination of the colloid and adenomatous. At the onset

of these cases the colloid element predominates and the thyroid enlargement is uniform, smooth and symmetrical, no adenoma being palpable. Later the character changes and the adenomatous element predominates. Coller found in an analysis of the pathological reports of five hundred goitres, 67.80% of adenomas and but 18% of colloids; Coller also reported in the Journal of the A. M. A. of May 31, 1924, a very interesting analysis of three hundred cases of adenomatous goitre. The patients were all above 20 years of age with a basal metabolic rate within normal limits. The analysis was made to determine what percentage showed tracheal compression, cardiovascular symptoms or psychoses. The patients were grouped according to age into decades from the third to sixth inclusive. There was a very definite increase from the third to the sixth decade in the percentage of cases found to have an enlarged heart or other circulatory disturbance. Coller mentions the usual increased occurrence of these conditions found with advancing age in persons without thyroid enlargement but does not believe the percentage would be nearly as high as in this group, and concludes these cases may have suffered a low grade toxemia over a long period of time or may have had exacerbations of hyperthyroidism in the past without symptoms severe enough to attract attention. It seems fair to assume from our present knowledge that a fairly large percentage of goitre cases will sooner or later show degenerative changes.

Fortunately we now know that the development of colloid and adenomatous goitres can be prevented by giving a proper amount of iodine to women during pregnancy and to children during the pre-adolescent period. We have a right to feel that this type of goitre will ultimately disappear but the problem of how best to supply the proper amount of iodine to all persons in the endemic goitre areas has not been entirely solved, nor have the people generally yet come to a realization of its importance. In Michigan the salt manufacturers are marketing a table salt containing .02% iodine which is

being extensively used and which we hope will ultimately reduce the number of cases in our state.

In the selection of goitre cases a history of past or present hyperthyroidism is of paramount importance. Fundamentally a toxic case at time of application should not be accepted and a past history of toxicity calls for a careful consideration of its duration, severity and the time which has elapsed since the disappearance of all symptoms. A case with such a history that had not been operated could hardly be considered because of the probability of recurrence. Toxic cases which have been operated with entire freedom of symptoms for a period of years, and who show no degenerative changes, are probably acceptable but it would seem good discretion to not even accept such cases without a rating. The company with which I am connected has had very little experience with goitre risks notwithstanding the fact we are located in an endemic goitre state. Until very recently we declined all goitre cases. Of late we have been accepting applicants under 25 years of age who have small goitres on 20 Year Endowment policies, provided the goitre is a uniform enlargement of the entire gland and not nodular. A goitre described by the examiner as limited to one lobe, or nodular, we consider adenomatous and rate 125%, even though the applicant is under 25 years of age. Beyond age 25 we rate both class of cases from 125 to 135 per cent, depending upon all the factors in the individual case. These ratings of course refer to applicants with small goitres which are not of sufficient size to have ever caused any pressure symptoms. We have never considered any cases of exophthalmic goitre, even after successful operation.

Dr. Ward—We have all enjoyed Dr. Hutchinson's discussion, and we will now hear from Dr. Bradshaw:

Dr. Bradshaw—Dr. Grosvenor in his admirable paper presented to this Society last year, reporting on goiter, stated that the experience of his Company showed a mortality of 159% on lives and 253% by amounts. In our Company we have had

a similar experience. An analysis of the issues from 1907 to 1920, exposed to 1923, gives a mortality of 162% on lives and 137% by amounts. On the other hand, we have had a very favorable experience in the group where the goiter has been partially or wholly removed by operation. These figures quite accurately verify the clinical reports of physicians who see many cases of goiter. Another point to be emphasized is that the majority of deaths has occurred in the age group 15 to 40—rather than a group of 40 and over.

I believe that this unfavorable experience is due to the fact that we have not understood what each type of goiter means clinically, how it is possible to recognize these types and what is most important, the pathological changes that goiters undergo during a period of 10 to 20 years. Dr. Carber has given us a very clear picture of these features in his excellent paper and has made suggestions by which it is possible to make definite rules of selection.

Part of the poor mortality is apparently due to the fact that the adenomatous type of goiter is so seldom diagnosed in its earliest stages. Dr. Frederick A. Coller in a recent paper entitled "The Morbidity of Endemic Goiter" says:

"The adenomatous goiter is diagnosed by the presence of nodules, the colloid goiter by its usual characteristics. Adenomas may well have been present but not palpable in the goiter called colloid, and with advancing age the increasing percentage showing adenomas is marked. One may fairly assume that the goiter called colloid up to the age of 25 is the same goiter called adenomatous at 45 with the change in the prominence of the component parts."

To show you the frequency of the adenomatous type of goiter, I quote from the same paper a pathological classification of 500 goiters showing the percentages of types in each group.

| | |
|-----------------------------------|-------|
| 1. Hyperplasia | 9.96 |
| 2. Colloid or Simple Hypertrophic | 18.00 |
| 3. Adenomatous Goiters | 67.80 |
| 4. Carcinoma | 1.25 |
| 5. Sarcoma | 0.66 |
| 6. Thyroiditis | 1.25 |

Analysing different types of goiters in another group of 993 cases, Coller found that at age 25 there were 66% colloid goiters and 15% adenomas while at age 44 there were 12% colloids and 73% adenomas.

With this clear presentation of the physical and clinical characteristics of goiter before us, bearing in mind the sequence of pathological changes that goiters undergo and the relative frequency of the adenomatous type, which so often leads to cardio vascular changes, together with the vast fund of information now available about the so-called goiter districts, we are in an excellent position to make a scientific selection of this medical impairment.

Dr. Ward—Dr. Pollard will continue the discussion.

Dr. Pollard—On approaching the discussion of Dr. Carter's paper on "The Present Status of Goiter," the thought of the very fine papers on the subject of goitre read before this Association by Dr. Rowley in 1917 and Dr. Harlow in 1922 comes to one's mind, and I am moved to wonder whether or not I can add anything of value to the subject, and yet the importance of the part played by the thyroid gland in the general well-being of the individual is so great that even at the risk of repeating many things which have already been said it seems to me it would be well to say a word regarding its functions.

McCarrison in his work on the thyroid gland gives the functions of the apparatus as four in number:

1. To govern the growth of all cells and sustain their functional activity.
2. To control calcium metabolism.

288 Thirty-Fifth Annual Meeting

3. As a katabolic stimulant facilitating the breaking down of exhausted cells and governing the elimination of the waste products of their disintegration.

4. To exercise a protective anti-toxic and immunizing action, protecting the body not only against the toxic products of its own metabolism but against invasion by the micro-organisms of disease and their products, and these it does by discharging into the blood stream and lymph a complex secretion which contains the active principles or hormones.

Kendall has stated that the real function of the thyroid is the production of thyroxin and the colloid secretion seems to be used as a storehouse for the iodine which thyroxin needs for its elaboration.

Plummer (Journal A. M. A. July 23, 1921) has also given this as the highly essential function of the thyroid.

We should thus consider not only what part the gland takes in the normal person, but what may be taking place or has taken place in that person when a change has occurred in the structure and functions, and should think of the symptoms distinctly referable to it, such as pressure symptoms, nervous symptoms, tachycardia, etc., and also in reference to the body, economy as a whole reducing its resistance and making more grave any other impairments which are found at the same time in the examination of an applicant. We know of its close association with the other glandular structures of the body or hormone-producing organs, namely; the adrenals, thymus, pituitary body, pancreas, liver and the reproductive organs and the governing functions it exercises over these organs.

It maintains the cellular nutrition of these organs and controls bodily growth and metabolism, by reason of this and to a certain extent in its impaired function, occur the cases of cretinism and myxoedema.

In its action on the adrenals it indirectly affects the blood pressure and the blood supply to all parts of the body.

For purposes of action as insurance risks the division of the different types into simple, adenomatous and exophthalmic seems best, though no hard and fast rule can be drawn as it is a well-known fact and should always be borne in mind that a simple goitre is potentially toxic and may become so at any time.

The action an insurance company should take in regard to its treatment of these cases has been determined frequently in the past by the personal opinion of the Medical Officer acting upon the application and it is hardly to be wondered at, as our statistics have been meagre, though in our own Home Office we are tabulating these cases (as I know many of you are) and in a short time should be better able to say with authority whether or not an applicant with a goitre or a history of one is a good risk to take and upon what plan.

A few years ago I wrote a number of the surgeons of the country who were doing a great deal of thyroid work and found their opinions so varied and based upon personal opinion largely that they were of no actual value. For instance, some of them thought that if in two years after an operation for exophthalmic goitre the patient was all right they would be good risks; others thought they would be after three years; and others said they never would be.

While the simple or colloid type produces no systemic effects, it may, by reason of its size and position, give rise to symptoms of a mechanical nature due to pressure on nearby structures, such as the trachea, great vessels, pneumogastric or recurrent laryngeal nerves, etc. This type occurs usually between the ages of 15 and 25 and as Plummer says usually disappears before the 25th year.

The adenomatous type is one of middle age, but usually shows some enlargement of the gland in early life. In this type Plummer says the average interval between the time it is first noticed and the onset of hyperthyroidism is 17 1/2 years.

The exophthalmic goitre usually occurs between 30 and 50

290 Thirty-Fifth Annual Meeting

and is much more rapid than the adenomatous type of toxic nature.

Lt. Commander Hayden (U. S. Naval Medical Bulletin, September, 1924) mentions the fact that these cases have a characteristic weakness and sinking in of the quadriceps muscles of the thigh and cannot, because of this, step up on a low stool or chair.

Should there be any question of the nervousness, tremor, tachycardia, etc., which occasionally occurs with simple goitre, being due to hyperthyroidism, the basal metabolism rate will prove which it is, as in simple goitre, the rate is unchanged or diminished while in true hyperthyroidism it is increased, especially so in the exophthalmic type.

In toxic adenoma, the cardio-vascular system is most affected, while in the exophthalmic goitre it is the nervous system.

When, therefore, we have before us a case showing an enlargement of the thyroid there are a number of questions which arise in our minds and the answers to which will guide us in determining our action.

First of all, what is the age of the applicant, when was the enlargement first noticed, how rapidly has it grown, what is its present size, has it given rise to any symptoms of pressure or of a toxic nature, is there a history of weakness or loss of weight and is exophthalmos present?

If an operation has been performed, what was the reason for doing it, what was its date, type and how much of the gland was removed, how long did convalescence take and has there been any evidence of a recurrence?

During the year 1922, there were received in our Ordinary Department 335,559 applications, and of these, 793 cases had goitre at the time of examination or gave a history of such a condition at some time in the past, with or without operation. There were also 86 cases in which goitre had previously been reported, but which did not give any evidence of such a

condition at the time of examination. According to sex these were distributed as follows:

| | |
|---------|-----|
| Males | 260 |
| Females | 533 |

The ages were as follows taken during five-year periods after 20 and under 50:

| | |
|----------|-----|
| Under 20 | 109 |
| 21 to 25 | 167 |
| 26 to 30 | 152 |
| 31 to 35 | 91 |
| 36 to 40 | 103 |
| 41 to 45 | 71 |
| 46 to 50 | 47 |
| Over 50 | 53 |

Of these cases, 198 were approved and 595 rejected, though some of the latter had other impairments which caused or contributed to the action taken.

There was, as will be seen, a preponderance of females (over 2 to 1) and most of them were under 30 years of age, the number occurring after age 40 being comparatively few.

All men, having thyroid enlargement, should be examined systematically for evidence of cardio-renal pathology (Brendel & Helm—Archives of Internal Medicine, January, 1919).

We should have a blood pressure reading in every case which has been operated upon, either for a simple goitre or any other type. Our practice at this time is not to accept any type during the first year after it has been operated upon, and the exophthalmic type not during the first three years after operation. From one to three years after operation the simple type is accepted at Standard to 150% rating and after three years at regular rates.

The exophthalmic between the 3rd and 5th years are accepted at 125 to 150 per cent rating and after 5 years at Standard to 130 per cent. When no operation has taken place, we do not take the exophthalmic cases or any case showing evidence of hyperthyroidism. The simple or adenomatous showing no evidence of growth for a year or two and no

292 Thirty-Fifth Annual Meeting

signs of pressure or other symptoms for that period are taken at Standard rates.

While our action in these cases is based upon our experience to a certain extent, yet it is not conclusive by any means, as our mortality statistics are, as yet, not sufficiently large to make us believe they may not have to be changed.

Dr. Ward—Dr. Eugene F. Russell of the Mutual Life will discuss this paper on Goiter.

Dr. Russell—The presentation of this paper on goiter by Dr. Carber at this time is very appropriate, as the mortality study of goitre, I believe, is to be made this winter by the joint committee of the Medical Directors' Association and the Actuarial Society. In this study, it is hoped that the classification as outlined by Dr. Carber will be followed as it is simple and scientific, and will prove whether or not the contentions of the clinicians are justified by medico-actuarial experience.

It will be interesting to discover if the actual mortality in goiter cases is favorable in the younger ages, gradually increasing and reaching a maximum after age forty, due to the fact, as many authorities contend, that 65% of the so-called colloid goiters are adenomatous. Likewise, it may be proven by this study that adenomatous goiters are not to be considered at standard rates, except perhaps at the younger ages, and then only on short term contracts; and that if taken at all, the mortality in such cases would be good in the early insurance years.

Incidentally, in the selection of these cases, it should be remembered that an asymmetrical goiter is usually adenomatous, and that the quiescent period lasts from 8 to 20 years. Mayos state 17 1/2 to 19 years, and that the age of activity usually occurs over forty. Hence, the age of an applicant with goiter is an important index to the insurability.

It is hoped that the mortality after thyroidectomy will also be investigated, and see if as the Mayos claim that 64% of

patients are cured six years after operation for exophthalmic goiter, and 83% two years after adenomata with hyperthyroidism.

The questionnaire as outlined by Dr. Carber is suggested as a guide to bring out the salient points, and to help the Medical Director in formulating an opinion. No case of thyroidectomy should be accepted unless a statement is obtained from the attending surgeon, and in addition to the questions as outlined by Dr. Carber, it should also be ascertained if all the adenomatous tissue was removed at the time of the operation, and if a microscopical examination of the tissue was made, as a small number of the adenomata show malignant changes.

Dr. Ward—Has Dr. Carber a word to say in closing?

Dr. Carber—I wish to thank these gentlemen for the discussion which I am sure has been constructive and instructive, particularly with regard to the physiology of the thyroid. With regard to thyroidectomy, I believe the greatest danger is that of recurrence, and practically all recurrences take place within the first three years. There is a small percentage, however, which occur between the third and fifth years, and I would place the period of acceptance of toxic goitres at five years. Non-toxic goitres can be safely taken at the end of two years.

Dr. Ward—Mr. James D. Craig, actuary of the Metropolitan Life Insurance Company, is going to present to you the subject of Disability Insurance, and it will then be discussed by some of our medical men. We are greatly obliged to Mr. Craig for this contribution, for I know it is a question which is puzzling a good many of you men, and I feel that Mr. Craig can help us very greatly in the solution of these problems. I take pleasure in introducing Mr. Craig:

DISABILITY BENEFIT IN LIFE POLICIES.

BY MR. JAMES D. CRAIG,

Actuary Metropolitan Life Insurance Company.

Disability benefits issued in connection with or supplemental to Life Insurance policies offer probably the most fascinating and complicated subject in the Life Insurance world today. Appearing for the first time in Life policies of the Fidelity Mutual in 1896, disability features have increased in popularity, until in 1923 approximately 223 Life companies doing business in the United States included some provision of this nature in their policies. In the beginning the disability benefit was designed only to prevent lapsation of Life Insurance and had consequently very limited scope. Its development in practice has probably been inspired and directed by competitive conditions rather more than by deliberate intent based on scientific principles and founded on proper experience; that is, the theory is following rather than leading in its evolution. It has even now had a very brief experience and may be regarded as still in the experimental stage.

The practical importance of the inclusion of this benefit in Life policies is evident from a study of the statistical tables in the Connecticut Insurance Report for 1923. The 33 Life companies there recorded show a premium income from Disability benefits of \$16,408,531, which is more than the entire premium income of 18 out of the 33 companies. The Disability benefits paid were \$5,606,380, which is more than 20 individual companies out of the 33 paid in death claims. If, therefore, all of these premiums were received by one company such company would be No. 16 as regards total premium income and No. 14 as regards the amount of claims paid. The Connecticut Insurance Report also gives statistics of 53 companies, both domestic and foreign, writing Casualty insurance. In general, of course, these are not the same companies as the 33 Life companies. The Report shows that in

1923 these 53 companies had a total net Accident and Health premium written of \$69,166,209, and total Accident and Health claims of \$32,365,423. While the benefits given under Accident and Health policies do not parallel those under Total and Permanent Disability benefits, we find that the Total and Permanent Disability benefit produced an income equal to 24% of the net premium written by the Casualty companies on their entire Accident and Health business, and that the claims were 17% of the Accident and Health claims of the same companies. This comparison with Accident and Health business of Casualty companies is made, not because Disability and Accidental Death benefits are Casualty insurance, but because they tend somewhat in that direction. The Insurance Commissioners have now before them a paper by the Commissioner of the State of Wisconsin, bringing forward the very definite proposition that Disability benefits are Health insurance, and raising the point as to whether the companies issuing Accident and Health insurance should not be protected from encroachment by Life Insurance companies issuing policies providing these benefits. In answer thereto the Travelers Insurance Company, as the company transacting the largest volume of Accident and Health Insurance of all companies in the United States, if not in the world, stated that it was not concerned with the argument of the Commissioner that Accident and Health Insurance companies should be protected from unfair competition, and that it had no occasion to fear evil results to its Accident and Health insurance from the incorporation by Life Insurance companies of provisions for Total and Permanent Disability benefits. Be that as it may, Life companies have or can have Accident and Health powers under statutes of all the states, as such powers have long been considered appropriate for exercise by Life Insurance companies. Moreover, the laws of many states which require different classes of insurance to be issued in separate policies, nevertheless provide that a policy

296 Thirty-Fifth Annual Meeting

of Life Insurance may be issued containing benefits against accidental injury or disease or disablement resulting from sickness. The Annual Statement for Life companies prepared by the Committee on Blanks and in conformity with the specific laws of many states which directly mention Disability and Double Indemnity benefits, requires the companies to separate and report, not only the premiums received for such coverage, but the claims paid and unpaid, as well as reserves held. The issuance of Disability and Double Indemnity benefits, therefore, whether considered as an integral part of a Life contract, or in the nature of Accident and Health insurance, is an established practice.

Developing, as it has, principally by competitive conditions, it is logical that the development should have been by a series of steps, each step making a stage in the evolution. At first there was the Waiver of Premium benefit, designed to keep the Life contract in force; then the Installment Payment benefit, reducing the face of the policy as paid, and finally the combination of keeping the Life policy in force by the Waiver of Premium plus installment payments which do not reduce the face of the policy and are known as annuity payments. After the development of the annuity benefit there followed a radical change in the definition of disability; heretofore benefit was paid only in event of *Total and Permanent* disability. Many of the new clauses do not require proof of the permanency of the disablement but pay for Total disablement provided such disablement has lasted for a stated period, usually three months. The time when the first annuity benefit would accrue has been reduced from six months after due proof has been received, until payments are now made either upon receipt of due proof or from the date at which Disability occurred. At the present time it is probable that over 90% of the companies provide the Waiver of Premium and Annuity benefits with a monthly income beginning either upon receipt of due proof or from date disability was incurred.

It is estimated that between 60% and 70% of the policies being issued to standard lives include Disability benefits and that this percentage would be larger were the benefit granted to all who apply for it.

It was stated above that these benefits tend toward Accident and Health insurance. The development, however, has been under the control and administration of men with Life Insurance training exclusively, and they are influenced, perhaps unconsciously, by the experience, practice and theories of Life Insurance, even when administering this different field. This has its advantages and disadvantages; it brings into prominence new experience, training and mental attitude, but in some cases fails to differentiate between principles of Life Insurance having to do chiefly with mortality and the principles of Disability insurance having to do chiefly with invalidity.

The new mental attitude is evident from the definition of total and permanent disability. Casualty underwriters had learned that "total disability" unqualified, may not mean unqualified total disability, that inability to perform "any duty" or to engage in "any occupation" may not mean "any and every duty" or "any and every occupation" and yet the inability to perform "all duties" may mean no more or less than the inability to perform "any duty." What constituted total and permanent disability was and is a question. At the last meeting of the Medical Section of the American Life Convention, Mr. Bates, in a discussion on Dr. Baker's paper on Applications and Claims, showed that courts had adopted literal, liberal or reasonable interpretations of what constituted Total and Permanent disability and he offered definitions of both total disability and permanent disability as follows:

"Total Disability is such as prevents the insured from performing his own occupation or the substantial duties thereof

298 Thirty-Fifth Annual Meeting

or any other occupation for which he is qualified by native ability, experience and education."

"Permanent Disability is such disability as will probably be permanent in the light of experience and medical science."

The obvious intention of the Total and Permanent Disability Clause is that the benefit shall be paid if the insured shall presumably be unable to earn an income for the rest of his life on account of being so disabled. The courts have held that "permanent" does not mean "perpetual" or "last forever" and that the "word permanent as thus used was not limited to incurable sickness, but imported such a state of disability as to preclude the hope that the deponent would be able to attend in any reasonable time." The disability must be something more than of a transitory nature; it must be disability which so far as can be reasonably determined will probably continue.

The last five words "reasonably determined will probably continue" indicate the difficulty in making hard and fast rules. The medical profession in clauses calling for Total and Permanent Disability will always have to reasonably determine whether the disability will probably continue, but the Life Insurance man comes forward with a practical suggestion as an aid and in the hope of eliminating controversy. This suggestion is that whenever disability has been total for at least 90 days, benefit will commence and be paid as long as disability lasts. The new mental attitude thus results in a definite attempt to provide a contract under which the liability can be plainly stated in such a way as to eliminate short temporary disablements, do away with many of the fine points for decision on the part of the medical director or lawyer, while at the same time giving the policyholder the benefit of the doubt.

The Life Insurance background again shows in the broad grouping of risks, the determination of the premium rate by ages, and the permanency of the contract. This is largely

due to the companies' actuaries. A flat rate at all ages, as is customary in Health insurance, was discarded and a definite attempt made to establish the cost at each age. These rates were generally applicable to all risks; occupational manuals calling for differentiation of rates by occupations were not prepared. It was generally felt that one large group of Ordinary risks could be secured upon which an adequate rate could be determined. Likewise, there was no thought of issuing a Disability benefit that could be cancelled should the experience prove unsatisfactory. The mental attitude of the Life underwriter, which naturally followed from his training, was that once the Life company issued a contract it should be uncancellable, even if the experience proved unsatisfactory.

While the mental attitude and training of Life Insurance officials has not changed, the experience is developing and medical directors, actuaries and other Life Insurance officials are studying it and seeking more light for their guidance in underwriting and administering the benefits. A clearer definition has been obtained. It has been definitely determined that when total and permanent disability is construed as such as can be "reasonably determined will probably continue" many cases will be admitted where the disability does not continue. In our company 17% of the admitted cases have subsequently recovered. The original table on which the premiums were predicated was based on a more stringent interpretation of Total and Permanent Disability and it is probable that companies are, therefore, admitting more claims for total and permanent disability than were expected. Now tables will have to be prepared, conforming more closely to current conditions; in fact the Actuarial Society of America has already appointed a committee to consider the present advisability of such action. Suffice it to say that the number who become totally and permanently disabled as compared with the expected by the present table is very materially higher

300 Thirty-Fifth Annual Meeting

at the younger ages than at the older. We have attempted to determine the number by age groups according to our very limited experience. The percentage of actual compared with the expected runs from 215% at ages 15 to 19, to 100% at ages 55 to 59. These rates are based on a benefit providing Waiver of Premium and \$10 a month per \$1,000 of insurance, with the first annuity payment six months from due proof of Total and Permanent Disability. When the payments commence upon receipt of due proof or from beginning of disability, lasting as total for three months, the rates will obviously be larger. The rate of disability, also the ratio of the estimated actual ages compared with the expected by Hunter's table, is shown in the following table:

Table Showing Rate of Disability on the Metropolitan Ordinary Policies With Waiver of Premium and Annuity Benefit: Also Percentage of Hunter's Table

| Age | Disability Rate | % of Hunter's Table |
|-------|-----------------|---------------------|
| 15-19 | .00110 | 215% |
| 20-24 | .00109 | 210 |
| 25-29 | .00107 | 199 |
| 30-34 | .00100 | 172 |
| 35-39 | .00100 | 142 |
| 40-44 | .00121 | 128 |
| 45-49 | .00154 | 116 |
| 50-54 | .00203 | 100 |
| 55-59 | .00351 | 100 |

A ratio of actual to expected ranging from 215% down to 100% conveys a definite impression, but the meaning of rates of disability running from 110 to 351 per 100,000 is not so obvious. Their full significance, however, should be clearly appreciated, not only by medical directors but by all connected with the Life Insurance business. They tell a very different story from what some might suspect. The premium charged is relatively small when compared with the premium charged for the Life Insurance benefits, and the impression may easily be created that Disability benefits are more in the nature of frills to a Life policy than a real and vital part thereof. A

study of the rates of individual companies will show that the actuaries consider the disability benefit as something more than a frill. In our company the Endowment at 85 rate at age 35 is \$24 per \$1,000; the Disability premium \$2.05, or 8½% of the regular premium. In the New York Life the premium for a Whole Life policy at age 35 is \$28.11; the Disability premium \$2.03, or nearly 7.22% of the regular premium. But even these ratios do not tell the whole story. The Disability benefit is generally conditioned upon disability occurring before age 60. A Whole Life policy provides for death occurring, not only before age 60, but thereafter as well. The difference between Life insurance ceasing at age 60 or continuing thereafter, is made manifest by the following table, which shows the number out of each 10,000 entrants at quinquennial ages who, according to the American Men Ultimate Table, attain age 60:

Table Showing Out of Each 10,000 Entrants the Number Attaining Age 60 and the Number Dying Previous Thereto According to the American Men Ultimate Table

| Age at Entry | No. Attaining Age 60 | No. Dying Previously |
|--------------|----------------------|----------------------|
| 20 | 7083 | 2917 |
| 25 | 7230 | 2770 |
| 30 | 7391 | 2609 |
| 35 | 7561 | 2439 |
| 40 | 7758 | 2242 |
| 45 | 8019 | 1981 |
| 50 | 8400 | 1600 |

Out of 10,000 who enter at age 20, 7,083 attain age 60 and 2,917 die prior thereto. If, therefore, Life insurance policies were issued agreeing to pay \$1,000 in the event of death occurring before age 60 the company issuing them would plan to pay 2,917 deaths out of 10,000 entrants; whereas if the policy issued provided for the payment of \$1,000 in event of death either before or after age 60, the company would plan to pay the whole 10,000 claims; in other words only 29.17% of the entrants at age 20 are expected to die before age 60 according to the American Men Ultimate Mor-

302 Thirty-Fifth Annual Meeting

tality Table. In computing a Life premium the fact that 70.83% will die after age 60 is taken into consideration and a considerable part of the premium is reserved for such claims. To say that a Disability premium, which provides a benefit ceasing at age 60, is 8 or 8½ per cent of a Life premium which provides a benefit for the whole of life, underestimates the comparative importance of the disability benefit.

The rates of disability when clearly understood and studied in connection with similar tables of rates of mortality gives the true facts, and eliminates any false impressions as to the frequency of disability. For our purposes the rates may be compared with the mortality rates of the American Men Ultimate Table, but at the present time most companies are experiencing mortality even more favorable than under this latter table, and comparison is, therefore, made not only with the American Men Table, but also with a table showing, after eliminating the first five years of exposure, the Metropolitan Ordinary experience during 1923. In making this table we have assumed that the average rate for each five age group could be taken as the rate of the central age. The experience has not been graduated, and shows, therefore, in its crude form.

Table Showing Mortality Rate Per Thousand According to the American Men Ultimate Table and Metropolitan 1923 Ultimate Experience, Compared with the Metropolitan Rate of Disability as Shown Above

| Central Age | Yearly Deaths per 1000 | | Annual Disability Rates per 1000 Metropolitan Exp. | Annual Disability Rate is of Annual Death Rate | | |
|-------------|------------------------|-------------------------|--|--|--------------|-----------|
| | Am. Men Table | Metropolitan Experience | | Am. Men Ultimate | Metropolitan | 1923 Exp. |
| 22 | 4.12 | 2.93 | 1.09 | 26 | 37 | |
| 27 | 4.39 | 3.05 | 1.07 | 24 | 35 | |
| 32 | 4.51 | 2.89 | 1.00 | 22 | 35 | |
| 37 | 5.12 | 3.87 | 1.00 | 20 | 26 | |
| 42 | 6.54 | 5.39 | 1.21 | 19 | 22 | |
| 47 | 9.18 | 7.45 | 1.54 | 17 | 21 | |
| 52 | 13.62 | 10.75 | 2.03 | 15 | 19 | |
| 57 | 20.69 | 15.71 | 3.51 | 17 | 22 | |

This table shows that if the numbers exposed were the same, the Disability Claims would range from 19 to 37 per cent of the Death Claims and on the basis of the American Men Ultimate Table from 15 to 26 per cent. These ratios, while they might seem high, are probably not as high as they will become, as the Disability rate was based on an experience under a benefit where the first payment was made six months after due proof of disability. When payments are made as of the date of proof, or from the date of Total disability considering three months of total incapacity as total and permanent disability, the disability rates will increase and the percentages such rates are of the corresponding death claims be correspondingly augmented.

With Disability rates per thousand such a large ratio of the death rates and with the ratio of the Disability premium to the Life premium unsatisfactory as a true criterion, what then is the real basis of comparison? In order to answer this question and at the same time show what part of a Whole Life premium is reserved for death claims after 60, the following table has been prepared, showing the net Whole Life premium based on the American Men Ultimate Table; the net premium for a Term policy expiring at age 60; the net premium based on our own table for a Disability benefit ceasing at age 60; the percentage of the Whole Life premium reserved for claims occurring after age 60 and the percentage the Disability premium is of the Term premium. The Disability premium has been adjusted to provide for the payment of total and permanent disability benefits as of the date of disability rather than six months from due proof, as it is felt that this benefit conforms more nearly to the current practices than does the benefit upon which our disability rates are based, also the rate of recovery has been introduced in its calculation.

304 Thirty-Fifth Annual Meeting

Table Showing the Whole Life Premium, the Term Premium, the Disability Premium, the Percentage of Life Premium above the Term, and Percentage of Disability Premium to Term Premium

| Age | Net Ann. Prem. 1000 W. L. Ins. | Term Ins. Expiring A. M. Ult. 3½ | Age 60 A. M. Ult. 3½ | Net Dis. Prem. for Disability oc- curring Prior to Age 60, Waiver | | % of W. L. Prem. for Claims Due at Date of Disability | Age 60 After Age 60 | % of Disability Prem. to Term Prem. |
|-----|--------------------------------------|---|----------------------------|--|----------|---|---------------------------|--|
| | | | | of Prem. Plus \$10 a Mo. | Reserved | | | |
| 20 | 10.72 | 6.30 | | 1.37 | 41 | | 22 | |
| 30 | 14.74 | 7.87 | | 1.64 | 47 | | 21 | |
| 40 | 21.75 | 10.83 | | 2.14 | 50 | | 20 | |
| 50 | 34.15 | 16.12 | | 3.19 | 53 | | 20 | |

When, therefore, a Whole Life policy is issued at age 20 and it is assumed that 70.83% of the claims will be paid after age 60; 41% of the premium is reserved for that later coverage and 59% is available for death claims occurring prior to age 60. When the Disability benefit is added, this Disability benefit costs 16% of this remaining 59%, and any benefit that costs one-sixth of the cost for the Life insurance benefit is not to be looked upon lightly.

The relative cost of the Disability benefit is more readily apparent in Group insurance. Under this form of insurance an employer insures the employees against death or disability during one year. The deaths within the year, as well as the disability claims, are therefore brought into contracts, not only with each other but also with the premiums paid. The disability benefit on group policies provides for the payment of the face amount of insurance and, based on amounts, the disability claims on Metropolitan Group policies for 1923 were 16.47% of the death claims. A table showing the percentage by age groups follows:

Table Showing Death Claims and Disability Claims Incurred During 1923 on Group One Year Term Policies.

| Age | Death Claims | Disability Claims | Percentage of Death Claims |
|-----|--------------|-------------------|----------------------------|
| 18 | 77,857 | 10,776 | 13.84 |
| 23 | 153,585 | 32,923 | 21.44 |

| | | | |
|-------|-----------|---------|-------|
| 28 | 180,042 | 50,357 | 27.97 |
| 33 | 195,836 | 32,984 | 16.84 |
| 38 | 242,038 | 44,400 | 18.34 |
| 43 | 328,072 | 41,570 | 12.67 |
| 48 | 383,188 | 59,505 | 15.53 |
| 53 | 387,149 | 51,397 | 13.28 |
| 58 | 391,759 | 61,350 | 15.66 |
| Total | 2,339,526 | 385,262 | 16.47 |

This table shows slightly different results from the previous one. Instead of the Disability benefit costing approximately 16% of the average premium for the Insurance benefit at each age it ranges from 27.97 at age 28 down to 12.67 at age 43, but it substantiates the general ratios given in the next to last column on page 302 and proves conclusively that the Disability benefit is an important feature no matter what class of business it is attached to.

CAUSES OF DISABILITY

What causes these rates of disability? One table showing cause of disability was published in Dr. Baker's paper. Others have been published in the TRANSACTIONS of the Actuarial Society. Such causes as tuberculosis, traumatism, cancer, and organic diseases of the heart affect both disability and death. There are others, such as blindness, which will produce disability but not death. Suicide, on the other hand, is death but not disability. Naturally there will be some border line diseases that produce death but may or may not produce disability claims, and if they do claims should be relatively few, as for instance, pneumonia. The records of 695 cases admitted by the Metropolitan for Total and Permanent Disability under the Annuity benefit have been classified by cause of Disability, and 4973 Ordinary Death claims paid by the Metropolitan in the first six months of 1924 have been classified by cause of death. These are shown in the following table grouped according to those diseases which produce both disability claims and death claims, and those which produce more specifically either disability or death claims.

306 Thirty-Fifth Annual Meeting

Table Showing Disability and Death Claims, Classified by Cause of Claim

| CAUSE OF DISABILITY OR DEATH | Disability | | Death | |
|--|------------------|------------------------|------------------|------------------------|
| | No. of Claims | Percentage of Total | No. of Claims | Percentage of Total |
| Tuberculosis | 355 | 51.1 | 562 | 11.3 |
| Traumatism & other external causes | 43 | 6.2 | 405 | 8.1 |
| Cancer | 16 | 2.3 | 525 | 10.6 |
| Organic diseases of the Heart | 11 | 1.6 | 583 | 11.7 |
| Total | 425 | 61.2 | 2075 | 41.7 |
| CAUSE OF DISABILITY | | | | |
| Insanity | 106 | 15.3 | | |
| Paralysis | 43 | 6.2 | | |
| Other diseases of Nervous System | 16 | 2.3 | | |
| Diseases of Spinal Cord | 11 | 1.6 | | |
| Blindness | 10 | 1.4 | | |
| Inflammation of the Brain | 9 | 1.3 | | |
| Diseases of Bones & Organs of Locomotion | 8 | 1.2 | | |
| Anaemia | 7 | 1.0 | | |
| CAUSE OF DEATH | | | | |
| Pneumonia | | | 470 | 9.5 |
| Chronic Nephritis | | | 303 | 6.1 |
| Cerebral Hemorrhage | | | 289 | 5.8 |
| Appendicitis | | | 160 | 3.2 |
| Influenza | | | 128 | 2.6 |
| Suicide | | | 149 | 3.0 |
| Diabetes | | | 69 | 1.4 |
| Respiratory diseases | | | 55 | 1.1 |
| Puerperal state | | | 52 | 1.0 |
| Other causes | 60 | 8.5 | 1223 | 24.6 |
| | 695 | 100. | 4973 | 100. |

The same objection arises against the comparison in this table as in the comparison of premiums, namely, the deaths included those occurring beyond age 60 while the disabilities are limited to those occurring before age 60. This combined with the newness of the disability benefit explains why the death claims from cancer are 10.6% whereas the disability claims are only 2.3%. Tuberculosis is the outstanding cause of Disability, with insanity second. In Death claims, tuberculosis is the second cause and insanity of practically no importance. This is the impression gained by comparing mortality statistics covering a large experience over the span of life with the very limited Disability statistics covering only a period of life.

The table shows that of the four causes of death organic diseases of the heart show the larger proportion, being 11.7% against 11.3% for tuberculosis. When, however, the deaths occurring after age 60 are eliminated and when deaths are considered only on policies that have been issued since the Disability annuity benefit was adopted, a very different story is told. Deaths from tuberculosis are twice as numerous as deaths from cancer, while for the four major causes common to both death and disability claims the death follow the identical order of the Disability claims, with tuberculosis first, traumatism second, cancer third and organic diseases of the heart fourth.

Table Showing Disability Claims and Death Claims Occurring Within Five Years from Issue and Under Age 60, Classified by Cause of Claim

| CAUSE OF DISABILITY OR DEATH | Disability | | Deaths | |
|--|------------------|------------------------|------------------|------------------------|
| | No. of Claims | Percentage of Total | No. of Claims | Percentage of Total |
| Tuberculosis * | 355 | 51.1 | 349 | 17.6 |
| Traumatism & other external causes | 43 | 6.2 | 204 | 10.3 |
| Cancer | 16 | 2.3 | 172 | 8.7 |
| Organic Diseases of the Heart | 11 | 1.6 | 131 | 6.6 |
| Total | 425 | 61.2 | 856 | 43.2 |
| CAUSE OF DISABILITY | | | | |
| Insanity | 106 | 15.3 | | |
| Paralysis | 43 | 6.2 | | |
| Other diseases of Nervous System | 16 | 2.3 | | |
| Diseases of Spinal Cord | 11 | 1.6 | | |
| Blindness | 10 | 1.4 | | |
| Inflammation of the Brain | 9 | 1.3 | | |
| Diseases of the Bones and Organs of Locomotion | 8 | 1.2 | | |
| Anaemia | 7 | 1.0 | | |
| CAUSE OF DEATH | | | | |
| Pneumonia | | | 192 | 9.6 |
| Chronic Nephritis | | | 60 | 3.0 |
| Cerebral Hemorrhage | | | 53 | 2.7 |
| Appendicitis | | | 102 | 5.0 |
| Influenza | | | 65 | 3.3 |
| Suicide | | | 69 | 3.5 |
| Diabetes | | | 19 | 1.0 |
| Respiratory diseases | | | 21 | 1.0 |
| Puerperal state | | | 47 | 2.4 |
| Other causes | 60 | 8.5 | 502 | 25.3 |
| | 695 | 100 | 1986 | 100 |

RECOVERIES

The definition of Total and Permanent Disability was that disability must be such as can be "reasonably determined will probably continue." In many cases it is thought the disability will probably continue but experience proves otherwise, and so, starting out to give one benefit, companies find themselves within a short time actually giving another. While the original disability was such as to render it improbable that the insured would again be active within a reasonable time, nevertheless recovery followed and quite often total and permanent disability resolves itself into total temporary disability. This is not emphasised with the idea of maintaining that only those cases should be recognized where disability will undoubtedly continue until death, but to bring clearly into prominence the character of the benefit actually given. While the general disability experience is of relatively recent origin and consequently limited, the recoveries from disability must naturally be more so, but it is significant that already two-thirds as many presumably total and permanent disabled cases have recovered as have died, the percentage of admitted cases being 17 for recovery and 26 for death. The following table shows 695 cases classified according to cause of disability, and also as to whether the insured died, recovered, or was still on disability up to Sept. 30, 1924.

Table Showing Classification of 695 Cases, Issues of 1918 to 1922 Inclusive, Where Disability Occurred Before Anniversary in 1923, According to Cause of Disability; Also Whether the Insured Died, Recovered or Was Still on Disability Sept. 30th, 1924

| | No. of Deaths | No. Recovered | No. on Disability | Total |
|--------------------------------|---------------|---------------|-------------------|-------|
| Tuberculosis (Pulmonary) | 121 | 61 | 164 | 346 |
| Tuberculosis (other varieties) | 2 | 2 | 5 | 9 |
| Cancer | 11 | 1 | 4 | 16 |
| Acute Articular Rheumatism | | 3 | 3 | 6 |
| Anaemia | 2 | | 5 | 7 |
| Other general diseases | 2 | | 3 | 5 |
| Inflammation of the Brain | 1 | 1 | 7 | 9 |
| Locomotor Ataxia | 1 | 1 | 4 | 6 |
| Diseases of Spinal Cord | | 2 | 9 | 11 |

| | | | |
|--|-----|-----|-----|
| Apoplexy | | 5 | 5 |
| Paralysis | 7 | 6 | 43 |
| Insanity | 20 | 15 | 106 |
| Other diseases of Nervous System | | | |
| Blindness | 3 | 4 | 16 |
| Organic diseases of Heart | 1 | 9 | 10 |
| Diseases of Bones & Organs of Locomotion | 3 | 1 | 11 |
| Traumatism | 2 | 5 | 8 |
| Fractures | 1 | 14 | 16 |
| External violence | 2 | 7 | 22 |
| Miscellaneous (less than 5 in any cause) | 1 | 3 | 5 |
| | 4 | 12 | 36 |
| | 184 | 119 | 392 |
| | | | 695 |

It is expected that a large number of totally and permanently disabled will die quickly, so that 184 deaths out of 695 admitted cases is reasonably in accordance with these expectations. If the admitted cases had been considered as 695, less 119 recovered, or 576 cases, the deaths in this short time would have been nearly one-third. Certain phenomena are recognized as natural, as for instance, no recoveries from blindness and a high death rate from cancer. Diseases such as insanity, blindness and traumatism, can be expected to give long durations of disability, while from tuberculosis 52% of the policies either cancelled quickly by death or recovery, but the remaining 48% are still disabled. Undoubtedly as the experience broadens and liberality in recognizing disability continues, the percentage of recoveries will materially increase. Recovery rates affect the experience on disabled lives in just the same manner as death rates. The factors determining the actual cost depend on the loss ratio actually incurred, and this of course can only be determined after the last disabled life has either died or recovered. Digressing a moment, it might be stated here that while most of the larger companies show a financial loss at the end of 1923 from disability, a large proportion of this loss is occasioned by setting up reserves, part of which may never be actually needed on account of recoveries.

310 Thirty-Fifth Annual Meeting

A rather definite comparison of cost of disability and mortality incurred during the early years of the policy and by different age groups, after allowing for both disabilities and recoveries, can be obtained, since the value of the disability benefit is about equal to the amount of the death claim. In publishing the American Men Table in the Report on page 166, Vol. II, the death rates by age groups and durations were given for certain causes of death. Similar rates of disability have been prepared from the Metropolitan experience and appear in the following table:

Table Showing Comparison of Death and Disability Rates for 10,000 Exposed for Total of All Causes, and Also for Tuberculosis and Insanity Respectively

| | Age | Total Claims | | Tuberculosis Claims | | Insanity Claims | |
|--------------------------------------|-------|--------------|------------|---------------------|------------|-----------------|------------|
| | | Death | Disability | Death | Disability | Death | Disability |
| First Policy Year | 20-29 | 28.4 | 9.1 | 2.3 | 5.8 | 0 | 1.1 |
| | 30-39 | 32.9 | 7.1 | 1.5 | 2.6 | .1 | 1.5 |
| | 40-49 | 43.4 | 8.3 | 1.5 | 2.2 | .7 | 1.8 |
| | 50-59 | 97.6 | 33.0 | .7 | .0 | .4 | 1.2 |
| Second Policy Year | 20-29 | 36.0 | 11.6 | 7.0 | 7.7 | .1 | .5 |
| | 30-39 | 41.2 | 13.6 | 5.0 | 7.1 | .5 | .5 |
| | 40-49 | 67.0 | 12.3 | 2.9 | 3.0 | .6 | 5.1 |
| | 50-59 | 125.9 | 24.2 | 3.8 | 0 | 1.1 | 8.2 |
| Third, Fourth and Fifth Policy Years | 20-29 | 38.9 | 12.5 | 10.2 | 7.5 | .1 | 1.8 |
| | 30-39 | 44.9 | 10.0 | 7.4 | 5.0 | .4 | 1.5 |
| | 40-49 | 68.4 | 18.9 | 4.4 | 5.6 | 1.0 | 2.1 |
| | 50-59 | 136.6 | 13.8 | 3.7 | 1.0 | .9 | 3.8 |

The table shows that in the first year the Total Disability claims are less than one-third of the Death claims, while the Disability claims caused by tuberculosis are nearly double the Death claims. The claims from insanity are practically incomparable. At first thought it may seem that a tuberculosis disability rate of twice the death rate was not comparable with the Metropolitan figures showing that tuberculosis gave 51% of the Disability claims and only 17% of the Death claims, but a little further analysis will show this reasoning is fallacious. In the age groups 20-29 the total Deaths were

28.4 and the Deaths from tuberculosis 2.3 or 8% of the total. In this same age group the total Disability claims were 9.1 and the claims from tuberculosis 5.8 or nearly 64% of the total, so that on this one age group the percentage of Disability claims from tuberculosis would be nearly 8 times the percentage that tuberculosis Death claims were of the total Death claims.

Referring to the second and third to fifth policy years, the Total Disability claims become a smaller proportion of the Total Death claims as the age advances, while the tuberculosis Disability claims tend to approach the tuberculosis Death claims. As would be expected, the Disability claims from insanity continue much higher than the Death claims from this cause. The increase in the Total Death claims from the second year to the third, fourth and fifth years is relatively slight, being from 36.0 to 38.9 for ages 20 to 29 and similarly at other ages, and it is interesting to note that Disability claims follow somewhat the same trend. With tuberculosis the case is somewhat different. There is a more marked increase in Death claims over the second year but not in Disability claims. This increase in Death claims for the third, fourth and fifth years substantiates the increase of the tuberculosis Disability rate in the second year. If Disability did not increase in the second year Death claims might not increase in the third, fourth and fifth years. While a more protracted experience will show more clearly the ultimate loss ratio from different causes of Disability, the present indications, with the limited experience, are that except for longer durations tuberculosis will always cause the largest percentage of claims and that the Disability rate will approach its ultimate just as quickly, if not more so than does the Death rate in Life Insurance, and that, this ultimate rate will be substantially incurred during the second year. This may seem contrary to some opinions. It may be argued that Disability claims in the second year are not nearly as high as in subse-

312 Thirty-Fifth Annual Meeting

quent years; but is this conclusion due to a study of incurred or admitted claims? The difference between these will be obvious from the following table, which shows the 281 Disability Annuity claims admitted by the Metropolitan in 1923 classified according to the year in which Disability actually occurred.

Table Showing Disability Annuity Claims Admitted in 1923 Classified According to Calendar Year in Which Disability Occurred

| Disability Occurring During the Calendar Year | Cases Admitted in 1923 |
|--|------------------------|
| 1923 | 97 |
| 1922 | 142 |
| 1921 | 27 |
| 1920 | 14 |
| 1919 | 1 |
| | 281 |

During 1923, then, 281 claims were admitted, of which 97 had become disabled in 1923, and 184 prior to that time. At the end of 1922 there were, therefore, 184 cases where disability had actually occurred on which claims would be submitted and approved in the next year, and it is perfectly obvious that there were more than 184 outstanding if we take into consideration those that will be approved in 1924 and 1925. The more liberal clauses now in use may result in claims being presented quicker.

Future experience should prove very enlightening, in these as well as in other respects. If the claims from tuberculosis diminish with advancing age, claims from degenerative diseases may continue to increase. What effect this will have it is hard to say. We know that deaths from cerebral hemorrhage produce 5.8% of the total, although considering deaths over age 60 only, the percentage is nearly 13. Organic diseases of the heart while causing 11.7% of the total deaths, produce nearly 23% of the deaths over age 60. We do know, however, that the chief problem of the day is the study and control of the early disabilities.

TUBERCULOSIS

Presenting as it does a large majority of our early claims, Tuberculosis seems to warrant special consideration. It occurs chiefly at the younger ages and accounts largely for our disability rate being over 200% of the expected according to Hunter's Table. At ages 20 to 29 the Metropolitan Disability claims from this cause were 5.8 for each 10,000 exposed in the first policy year, 7.7 in the second year, 7.5 in the third, fourth and fifth years. Is this reasonable? Have we been led to believe that we had a comparatively high disability rate from Tuberculosis because the Total Disability rate used as a measuring rod was inadequate, or is the basic table reasonably correct and has the company been imposed upon? What is a reasonable disability rate from Tuberculosis and how many claims should a company operating with a view of service to its policyholders be expected to pay? What statistics on Tuberculosis are available? The experience on Group Life policies paying the face of the policy in installments should total and permanent disability occur, may be of value. These policies are issued on the working men of the country. When an employer pays the premium all employees are covered; when the employees contribute at least 75% must be insured. No medical examination is required but the insurance is written according to a formula which precludes individual selection. Here we start with a group somewhat akin to the general population; everybody is working and disability claims from Tuberculosis arise from those who, subsequent to the issuance of the policy, become incapacitated from that disease. During 1923 the Metropolitan had \$410,000,000 of insurance exposed on Group Life policies. As shown earlier, the total disability claims were \$385,262, or 16.47% of the death claims. Of the \$385,262 disability claims \$133,872 were caused by Tuberculosis, and, classifying by ages, we find that for different age groups the disability claims from Tuberculosis per each 10,000 were as shown in the following table:

314 Thirty-Fifth Annual Meeting

Table Showing Disability Cases Arising From Tuberculosis per 10,000 Exposed in Different Age Groups

| Age | Claims per 10,000 Exposed |
|-------|---------------------------|
| 20-25 | 3.8 |
| 25-30 | 5.3 |
| 30-35 | 1.8 |
| 35-40 | 2.2 |
| 40-45 | 4.4 |
| 45-50 | 1.8 |
| 50-55 | 2.4 |
| 55-60 | 5.7 |
| Total | 3.3 |

No attempt has been made to smooth these rates and they are naturally somewhat irregular on account of the smallness of the exposure. The rate of 5.3 per 10,000 at ages 25 to 30 is lower than the first year's rate shown on medically examined lives where the policies contained the Annuity benefit. In both cases payment did not commence until six months from disability or proof thereof, so that to this extent the figures are comparable.

In addition to Group Life policies the Metropolitan issues Group Health policies under which indemnities are paid after the first week of incapacity. Unfortunately this business is not classified by ages, but during 1923 there were 28,380 years of life exposed under such contracts and 20 cases of incapacity, where the original diagnosis showed Tuberculosis, were incurred. Sometimes the first cause of disability is other than Tuberculosis, but subsequently the disability develops into Tuberculosis. We have been unable to look through the experience in the short time at our disposal to determine the number of these cases; suffice it to say that the claims during this year averaged 7 for 10,000 of exposure. On 14,696 years of exposure under Personal Accident and Health policies the Tuberculosis claims were at the rate of 8 per 10,000. This gives such figures as we have avail-

able for Tuberculosis on insured lives, but is the general trend of the Tuberculosis rate different from this, and if so will claims ultimately approach such general rate? Tuberculosis has been studied and you are all probably familiar with what is known as the Framingham Demonstration. As a result of the investigations made in Framingham, Mass., and elsewhere, it is generally conceded that in a city of 100,000 people 1000, or 1% will be active Tuberculosis, while another 1000 will be arrested cases, that of the 1000 active cases 8 to 10 per cent will die within the year and 5 to 15 per cent become arrested during the year. If the active Tuberculosis cases remain at about 1000 and if 8% of these die and 5% become arrested then 13% must occur as new cases to offset those deaths and recoveries. If 10% are taken as died and 15% as being arrested, then 250 out of the 1,000 cases cease to be active Tuberculosis cases, and 250 new cases must develop to keep the 1,000 constant. This means that out of a population of 100,000 from 130 to 250 will be afflicted with Tuberculosis each year, or on our basis of 10,000 from 13 to 25. This does not take into consideration the age distribution, and unfortunately statistics on this basis seem to be wanting. We do know that in Framingham 42% of the cases were between ages 20 and 44.

Another investigation of the incidence of Tuberculosis might be found in the General Survey of Communicable Diseases in the A. E. F. as appearing in the "Military Surgeon," October 1921. This survey shows that from July 1917, through April 1919, there were 4,201 cases of Tuberculosis of all kinds reported. Of these 2,113 were given as acute Miliary Tuberculosis, 102 as Tuberculosis of Bones and Joints, 735 cases of Incipient Pulmonary Tuberculosis and 1,251 cases of Chronic Pulmonary Tuberculosis, among the Expeditionary Forces; that the monthly incident rate of the group of Tuberculosis Pulmonary affections per 100,000 strength ranged from 111.8 in Jan., 1919, to 15.5 in Nov.,

316 Thirty-Fifth Annual Meeting

1918. The figures for the first four months of 1919, being the last figures given, were as follows:

Table Showing the Monthly Incidence Rates of the Group of Tuberculosis Pulmonary Affections in the A. E. F. per 100,000 Strength During the First Four Months of 1919

| Month | Rate per 100,000 |
|----------|------------------|
| January | 25.1 |
| February | 26.6 |
| March | 29.9 |
| April | 22.8 |

The rates for the last four months are given as probably indicating nearer to the true rate. Previous impairments had been more or less weeded out; the men as a whole were young, living an active outdoor life and subject only, apparently, to the extra hazard of gas.

The Survey states:

"It was thought that the damage as to the upper respiratory tract, to the bronchi and to the pulmonary parenchyma itself, resulting from exposures to various of the toxic gases used in battle, would predispose to tuberculosis or tend to develop into an active stage quiescent foci, which might have existed prior to the gassing. No evidence of any predisposition to tuberculosis or unusual incidence of tuberculosis among convalescent gas cases has been discovered so far."

For our purpose as presenting the number of cases of tuberculosis that are incurred in the course of a year by healthy men at young ages living outdoor lives these figures might indicate the normal incidence, and if the rate approximates 2 1/2 per 10,000 lives within a month this is equivalent to 30 a year.

Looking at the subject from another aspect, the Metropolitan has approximately 20,000 male employees; each year all these employees are medically examined in the hope of detecting disease and forestalling preventable sickness. It would seem that once this group was examined and steps taken to remedy existing impairments, the group might be looked upon as a reasonably healthy one and the impairments

found on the next examination would approximate closely those that had developed within the year, but out of these 20,000 men the Metropolitan recommended that 74 go to the Sanitarium for Tuberculosis in 1922, and that 89 go in 1923. This is at the rate of 37 and 44.5 respectively for each 10,000 men.

It is hardly to be expected that the incidence of Tuberculosis discovered by diligent search will be the same as Tuberculosis claims under Disability benefits where the claims are not submitted until disease has become more advanced, but these figures are given in order that all obtainable information be made available on the subject and the possibilities involved be clearly realized.

EARLY DISABILITY AND UNDERWRITING PRACTICES

If a diligent^{*} search for Tuberculosis finds 44.5 cases per 10,000 within the year, while Group policies with a six months' waiting period experienced at the younger age groups but 5.3 cases, and various other statistics range between these figures, then one of the practical problems involved in the underwriting would seem to be the detection of cases offering possibility of early claim. For this purpose we made a careful study of a number of disability claims, subdivided according to whether the insured had died, recovered, or was still apparently totally disabled. A very careful study of the application in face of later developments failed to discover any definite detailed family history or personal history which would seem to justify declining the case originally; yet out of 50 cases of disability where death or recovery had not occurred 9 cases of disability from Tuberculosis had occurred within one year of issue, 4 within six months and 2 within three months. Out of a total of 16 cases, 11 were caused by Tuberculosis where underweight existed running from 7 to 25 pounds. The family record was of no significance at all

318 Thirty-Fifth Annual Meeting

while such history of respiratory diseases as appeared was not of a character to invite underwriting consideration. On these 16 cases, the Tuberculosis not only started early, but gives promise of long duration, as on two of the cases we have now been paying five or six years, on 10 of them from four to five years, and on 4 from three to four years. A consideration of these cases in connection with race and low blood pressure, etc., was not undertaken on account of the small number, but we would commend these investigations to your further study. In 9 cases, Insanity was the cause of disability, and on six of these the disability occurred within fifteen months of issue and has continued to date. No history of any significance appears, except nervousness in one case three months from issue, with no detail as to severity, cause or duration. The quick occurrence of disability suggests possibly both adverse selection and malingering. Underwriting remedies for these factors do not appear from the application. It is possible that such remedies might be found in more thorough examinations with more particular regard to lung conditions and more searching inquiry as to previous personal history. It might be that inspections would be useful in this regard. Is it possible that the medical examination made for determining an applicant's eligibility for a Life policy does not specifically consider the dangers of disability? Is it possible for medical examiners by more careful examination of the lungs and the build, associated with color of face, appearance of eyes and evidence of wastage, to detect incipient tuberculosis? In view of the early appearance of Tuberculosis after examination in underweights at younger ages, must the lay underwriters save the situation by denying disability insurance to all young underweights, or can the medical men find a means of stopping the poor risks while permitting good risks to enjoy the protection of such insurance? Can the examiners be led to think seriously of mental as well as physical conditions in the course of their

examinations, having in mind the probability of Insanity? Can they detect the weak-minded, the neurotic, the markedly abnormal or ill-balanced mentally, in the course of examinations? Accident and Health underwriters often find that inspections reveal something in the history along these lines which the applicants are seeking to conceal. Can it be impressed upon medical examiners that more searching inquiry might be made and more careful consideration given to the drawing of conclusions? Are examiners too hurried, too superficial?

In the Economic World of September 27, 1924, Mr. V. R. Smith, Actuary of the Federation Life Association, Toronto, writing on "Life Insurance Without Medical Examination in Canada," says,

"So far as latent tuberculosis is concerned, how often is it discovered by the doctor, who himself is quite ready to admit that it is practically impossible to detect an impairment of this character in the time usually given to the average insurance examination? Accordingly the companies felt that while these risks might be accepted in greater numbers without medical examination, yet the number would not be greatly increased."

In considering the practicability of having a medical examination detect the marks of past suffering or incipient disease, which will shortly present a disability claim, what rules should be kept in mind in examining an applicant? If incipient disease exists the applicant seeking insurance can be expected to conceal it from the company, and the first and most difficult problem is the determining of such impending sickness. An illustration of this would be an insured who procured a policy in August and in September went to the country for several weeks on account of nervous breakdown; investigation showed a similar attack in the preceding May when, by the advice of a physician, he went to Atlantic City for a period; yet the true condition was concealed and not

320 Thirty-Fifth Annual Meeting

discovered by the examiner. Another applicant engaged in a seasonal occupation and likely to have "off-season" sickness, plans a trip in dull times and makes it appear that the trip is required by the state of his health. Again, an applicant contemplates retiring from business, and feeling that he can pass a good examination now, nevertheless expects a complete breakdown after getting the policy. Our company issued what is known as a non-cancellable Accident and Health policy to an applicant under date of December 14 after a careful medical examination. On December 29 the insured became totally disabled on account of physical and mental exhaustion and took a trip to Hot Springs. Investigation revealed that about a year previous he had an attack of neuritis and was laid up a couple of weeks; that a few months later he had considerable treatment and examination in an unsuccessful effort to locate a focal infection responsible for his poor health. Another insured took a policy in September 1922 and sold out his business on October 1 of the same year on account of poor health; he became disabled with appendicitis on November 14. Investigation showed an operation for hernia about one year previous and not disclosed.

Aside from this it is of great value to take precautions that the monthly benefit from all sources during disability will not exceed the earnings while in good health. All companies do not insert in their applications a question calling for amount of accident and health insurance together with life insurance carrying disability benefits; but such information is considered of the utmost importance in our office. One insured met with a serious sickness which resulted in disability benefits for a certain period. The insured felt that by selling his business and supplementing his investment income by the disability benefit he was perfectly justified in spending an indefinite period touring the country by motor and in general enjoyment. An illustration of how a man contemplating retiring from business may load up with disability and accident insurance,

was clearly brought out in a particular case recently. In 1922 application was made for non-cancellable Accident and Health insurance with weekly indemnity of \$75. Applicant was examined and recommended by the examiner as first class. The record showed that he carried a large amount of Life insurance with the company, that in 1921 he applied for more and was rejected as a poor moral risk. The application for Accident and Health insurance was declined, but later the case was reopened; he submitted statements and documents in defense of character and moral standing; the application was finally approved and policy issued. On the strength of it he procured large additional amounts from other companies, immediately after which the insured was driving alone in broad daylight on a smooth wide thoroughfare, and without known cause ran his car off the road down an embankment. The windshield was broken and he sustained cuts around the arms and legs and a cut on the cornea of the eye. His claims aggregated a very large sum. How good that eye was before the accident we do not know.

With these facts in mind, could more information be secured in the application? Might there not be questions asked somewhat as follows:

Whether any application previously made for Accident and Health insurance has been declined, postponed, withdrawn or issued on other than the plan applied for.

Whether any policy of Accident and Health insurance has been cancelled, rescinded, or renewal refused.

Whether the average weekly income from the occupation exceeds the aggregate disability benefit provided under all policies of Accident and Health insurance, or Disability Provisions of Life policies carried.

Whether there is any history of epilepsy, fits, vertigo or dizziness.

Whether the applicant has ever suffered from any ailment

322 Thirty-Fifth Annual Meeting

or disease of the skin, bones, glands, ears, eyes or teeth and gums.

It is not uncommon to have a question covering the first part of this last question, but it might be well to include a history of ailments of the teeth and gums as quite often serious disabilities can be traced to neglected diseases of those parts.

The benefit we are insuring is a substantial one, the company is bound on the risk for many years—possibly through many sicknesses. Such insurance must be written with care. The medical examination and the inspection must be minute and more and more attention devoted to incipient diseases, moral hazard, and to concealed facts. If a company is fortunate enough to have an Accident and Health record in its office it should be consulted for the past history of sickness whenever disability insurance is applied for, and if the record indicates any reason why an Accident and Health policy should not be issued it would be wise to refrain from granting the disability benefit.

Let me remind you that we are in business to pay claims. Our training and ambition prompts us to pay all legitimate claims quickly; but as regards Health insurance, Sir Alfred Watson once wrote that under contracts containing identical phraseology the company with the largest assets would pay the most claims, and Life insurance companies pre-eminently have the largest assets and will be expected to pay the most. The control of the business is largely managerial, and reduction in amount of benefits or lengthening of the waiting period will have instantaneous effect upon the disability rates. In like manner, an increasing benefit or reduction in the waiting period, or adoption of more simple rules, will be reflected in the losses. Total and Permanent Disability is elastic and in the last analysis will be just what we make it. Liberality with careless examinations and poor underwriting will be expensive, while careful investigation can produce satisfactory results.

One more aspect of this subject. Every sickness table that has been developed during recent years has shown a higher sickness rate than the previous one, while practically every life table has shown a lower death rate. If companies pay more in disability claims will the tendency be to have early disabilities submitted; and will the payment of a benefit thereon assist the policyholder to restored health or to attain longer life? Every year death is postponed the interest on the principle is earned, and if this were applied against the disability payments the net disbursement would be materially reduced. If a saving in mortality follows from a loss in disability does not the good of humanity demand that this loss be incurred, provided it is not excessive? Just where the mean is we do not know. We would not say that the reduction in mortality is caused entirely by the disability payments, but undoubtedly the patient can secure better care with the aid of such payments. Probably no association is better qualified than is yours to determine the correlation of the one benefit with the other.

In concluding I may say I have unhesitatingly taken advantage of whatever publications were available and have relied on the memorandum submitted to the Seattle meeting of the National Convention of Insurance Commissioners by the Equitable Life, Mutual Life and N. Y. Life. In addition I have availed myself of the services of the Metropolitan Staff, and am indebted to our Fourth Vice-President Stewart N. LaMont and our Assistant Medical Director Dr. Christiernin for their studies and suggestions; to our Statistician, Dr. Dublin, for various figures, and to all my associates in the Actuarial Division.

Dr. Ward—I will now call upon our newly elected second Vice President, Dr. W. W. Beckett, who is going to discuss this paper.

Dr. Beckett—The Pacific Mutual Life Insurance Company of California in its Accident Department, issues disability in-

324 Thirty-Fifth Annual Meeting

surance both on an examined and a non-examined basis. This refers to temporary disability benefits as distinguished from the Permanent Total Disability Benefits found in Life Insurance Policies. All applicants for the Permanent Total Disability are medically examined, and the experience under such policies is not referred to in this memorandum. Disability Benefits on examined risks are issued in conjunction with our Company's Life Insurance Policies, and the medical examination for the Life Insurance Policy constitutes the medical examination for the disability benefits. In addition, the Company issues what is called the Non-Cancellable Disability Policy, and these risks are all medically examined. If the Non-Cancellable Policy is applied for concurrently with a Life Insurance Policy the examination for the Life Insurance Policy constitutes the examination for the Non-Cancellable Policy, but if no Life Insurance is applied for a separate medical examination is required for the Non-Cancellable.

The experience on the temporary disability benefits issued in conjunction with Life Insurance Policies is kept by the Company on a premium and loss basis rather than on an exposure basis. On a premium income of \$2,750,000 for the disability benefits issued in conjunction with Life Insurance Policies (medically examined risks) we find that our accident indemnity claims are 32% of the accident indemnity premiums, and the sickness indemnity claims are 46% of the sickness indemnity premiums; the combined accident and sickness indemnity loss ratio being 40.7%.

Similar insurance is issued through the Commercial Division of the Accident Department under regular Accident and Sickness Policies. Risks for this form of insurance are not medically examined. On a premium income of \$5,800,000 we find that the accident indemnity claims are 43% of the accident indemnity premiums, and the sickness indemnity claims are 53% of the sickness indemnity premiums; the combined loss ratio being 47%. This loss ratio is produced on premiums

which are supposed to be loaded sufficiently to cover the absence of a medical examination. The experience shown indicates that the medically examined risks are more select from a disability loss viewpoint than are those which are written without a medical examination, notwithstanding that our company is more liberal in the settlement of claims in connection with life risks than those taken on the Commercial Plan.

The loss experience on the Company's so-called Non-Cancellable Disability Policy, which is issued on the basis of a medical examination, is practically parallel to that of the examined risks, with the exception that the moral risk creates an additional hazard. This is probably accounted for by the fact that the amount of Non-Cancellable disability insurance issued is larger than the amount of disability benefits issued in conjunction with Life Insurance, as the amount of Life Insurance applied for determines the amount of disability benefits which the Company will issue.

Our Company's experience with its Disability Insurance has brought out several interesting and vital facts. One is that an applicant entitled to Life Insurance is not necessarily entitled to Disability Benefits. We find that certain risks will live out their expectancy, but that they do not remain in good health during this entire time, and that their risk will be productive of a claim for disability benefits.

We find, further, that tuberculosis is the greatest single cause of disability, and this applies to medically examined risks as well as to those accepted without a medical examination. Certain rules for the acceptance of Life Insurance, which have become recognized in Life Insurance underwriting, have been found to be non-applicable to the underwriting of disability insurance, especially as affects the tuberculosis exposure.

A history of tuberculosis in the family must be carefully and thoroughly investigated, and the present and past physical condition of the applicant taken into consideration before disability insurance can be issued. Two cases of tuberculosis in

a family history should bar the issuance of disability benefits. One case of tuberculosis in a family, where death occurred from this cause at an age older than the applicant who is applying for insurance, should be looked upon with suspicion. One case of tuberculosis in the family, even at an age younger than that of the applicant, if the applicant himself is under normal weight and shows a history of repeated respiratory tract trouble, should be weighed very carefully before acceptance.

Experience has shown that a personal history of nervous breakdown, even though it occurred many years past, is practically a bar to disability insurance, although not necessarily a bar to Life Insurance.

One other feature which is proving an important factor in claim experience is syphilis. A personal or family history of any condition which might lead the examiner to suspect syphilis should be gone into extremely carefully before the issuance of disability insurance.

In issuance of large lines of disability insurance the moral hazard must be carefully considered. With the many forms of disability coverage now being sold it is possible for an applicant to secure insurance which, although possibly warranted by his income, may lead to an excessive and an unfair claim in the event of disability. The question of the amount of insurance carried in the issuing company and also in other companies, must be considered in connection not only with the applicant's present earning power, but with his prospects for the future. This applies particularly to men whose income is on a commission basis and may at present be large, but whose fortunes may change at a moment's notice under conditions possibly leading to nervous troubles and a long claim, lengthened and made more difficult to handle by reason of the large amount of indemnity carried.

Dr. Ward—Dr. Grosvenor of the Travelers will continue the discussion.

Dr. Grosvenor—We are indebted to Mr. Craig not only

for the interesting information he has gathered from various sources on the permanent and total disability provision and the splendid way in which he has marshalled his facts, but also especially for the new experience he has contributed. The experience on this provision in even the largest companies is so inadequate in itself that any additional information should be fully appreciated. The paper is so excellent that there is little, if anything, to criticise.

The theory of a permanent total disability provision in contracts of life insurance is a good theory. Continuance of life insurance protection is predicted upon the ability of the insured to make payment of the premium. If by reason of bodily injury or disease an insured shall become permanently and totally disabled to earn income, it is, of course, eminently desirable that the life insurance contract should be continued in force for the protection of the beneficiary. The earlier protective provisions of this kind maintained the insurance in force by waiver of further premium payments upon proof of permanent total disability. Later on, it was realized that this insurance protection might have to be sacrificed by loans or sales in order to provide means for the support of the insured and the members of his family during such disability and then the provision was broadened out so that the amount of the insurance could be preserved for the beneficiaries and a fixed monthly income of so many dollars for each one thousand dollars of insurance provided for the support and maintenance of the insured and his dependents.

With the principle that benefits of this kind should be limited to cases of permanent total disability and should not be payable for temporary disabilities we are in full accord. But for the integrity of the experience and the development and maintenance of right premium charges permanent total disability benefits should be restricted to bona fide cases of permanent and total disability. A company should not in order to avoid the obligation to investigate and determine the

merits of the claims as presented follow the so-called line of least resistance by paying indemnity for a short period for a disability which manifestly will not be permanent and total. The laws whether dealing directly with provisions for permanent and total disability or those which indirectly recognize such provisions by exempting them from the operation of the standard provision law, all clearly contemplate that there shall exist a condition of permanent total disability. We realize that it is not always an easy matter to determine that the disability alleged will be permanently total but difficulties as great as this are presented in the every day experience of insurance companies with regard to all kinds of insurance losses.

After the death of an insured, of course, it is a matter easy to decide whether or not during the period between a date prior to his death and the date of his death he was permanently and totally disabled but not always so easy in advance of the death to determine whether a disability commencing upon a given date will continue throughout the life of the insured or not. But these decisions must be made upon the best evidence obtainable in advance and the experience of the companies which furnish such benefits, as well as the experience of more than half a century of the companies which issue accident and sickness insurance, some of which cover permanent total disability, furnish a very good indication that the difficulties are not insurmountable and that, by and large, adjustments of the claims can be made with fairness to the insured and without sacrifice of reputation on the part of the company.

Historically Mr. Craig's paper may be enlarged upon. He refers to the origination of the clause by the Fidelity Mutual Life Insurance Company in 1896. This clause was decidedly complicated, considerable extra premium was charged and it bears little resemblance to other past or present clauses. Looking back to the year 1900 I think it safe to say that very

few insurance men at that time appreciated that there was such a thing as a disability provision issued. In 1904 the Travelers got out the first straight, premium waiver, permanent disability clause which centered the attention in life insurance circles to the possibilities of this feature now so generally adopted by other companies as well. The coverage of the clause has been largely improved upon and all disability contracts now contain the basic premium waiver. It is true that before that time both abroad and in this country some of the fraternal organizations gave permanent disability benefits but this was the first adoption of direct benefits for permanent and total disability with premium waiver.

A few points in further explanation of some of Mr. Craig's statements may be noted. He says that "the theory is following rather than leading" in the evolution of this provision. This is true especially in that no adequate experience is even yet available. "It, however, does not apply to the mathematical theory which has been developed as needed. In fact, all the essential theory except on very complicated benefits was given before the Third International Congress of Actuaries in 1901 by E. Hamza in a paper entitled "*Note sur la theorie mathematique de l'assurance contre le risque d' invalidite d'origine morbide, senile ou accidentelle.*"

Further light may be thrown on the growth of the premiums on this business by noting that the premiums received by all the companies in the country in 1923 amounted to about \$20,000,000, which was about double the corresponding premiums received in 1920 and about 40 times those received in 1913.

Mr. Craig appropriately calls attention to the difference in underwriting practice on the disability provision as compared with accident and health insurance, calling attention to the lack of differentiation of applicants as to occupation. To avoid misunderstanding, possibly reference should be made to the fact that the disability provision is not given or is limited

330 Thirty-Fifth Annual Meeting

to prospects engaged in various occupations and that there appears to be a tendency now to more carefully underwrite this provision in this respect.

Mr. Craig points out the importance of permanent total disability when measured with the probability of death on life contracts running to age 60. The data of the American-Canadian Mortality Investigation more clearly illustrates this. From it a comparison may be made of the probability of death from leading causes prior to age 60 at various ages at issue with the probability according to Hunter's table of permanent total disability during this period. For example, the probability of death from tuberculosis before age 60 on contracts issued at age 20 is 3.5%; whereas the probability of permanent total disability during the same period is 3.9%. At age 35 at issue the ratio for tuberculosis is 1.8% and the probability of permanent total disability, any cause, is 3.6%. It will thus be seen clearly that there is more chance of an applicant becoming totally and permanently disabled prior to age 60 than that he will die before age 60 of any one given cause.

This fact is shown in a somewhat different manner by the table below in which is given the relationship between the probability of death from any one cause prior to age 60 as measured against the permanent total disability which in this case is fixed 100%.

Death and Disability Probabilities Compared

Chance of Death Before Age 60, and 65, From Certain Causes Expressed as a Percentage of the Chance of Permanent Total Disability From All Causes Prior to the Same Ages

| Cause of Death | Prior to Age 60 | | | Prior to Age 65 | | | |
|----------------------------------|-----------------|--------|--------|-----------------|--------|--------|--------|
| | Age at Issue | 20 | 35 | 50 | 20 | 35 | 50 |
| Permanent Total Disability | | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% |
| Bright's Disease | | 74.2 | 77.5 | 67.6 | 69.9 | 69.7 | 61.2 |
| Organic Heart Disease | | 58.9 | 62.6 | 57.2 | 62.5 | 63.5 | 59.6 |
| Apoplexy and Cerebral Hemorrhage | | 51.5 | 57.2 | 61.7 | 56.3 | 58.8 | 59.8 |
| Pneumonia | | 55.8 | 51.5 | 43.1 | 48.1 | 43.6 | 36.5 |

| | | | | | | |
|--------------------------|------|------|------|------|------|------|
| Cancer and Other Ma- | | | | | | |
| lignant Tumors | 41.1 | 43.7 | 44.6 | 39.3 | 39.8 | 38.1 |
| Tuberculosis | 88.6 | 50.0 | 22.8 | 60.8 | 33.7 | 15.8 |
| Accident | 59.5 | 48.1 | 34.5 | 42.3 | 32.8 | 21.5 |
| Pericarditis and Angina | | | | | | |
| Pectoris | 25.1 | 27.7 | 26.8 | 26.1 | 27.1 | 25.8 |
| Diseases of the Arteries | 13.7 | 15.5 | 15.2 | 19.7 | 20.8 | 21.5 |
| Suicide | 31.7 | 30.5 | 27.1 | 22.5 | 20.3 | 15.5 |

I wonder if the full significance of the following statement of Mr. Craig is realized: "Recovery rates affect the experience on disabled lives in just the same manner as death rates." The Hunter's Disability Table used as a measuring rod with which to compare experience was based upon experience that probably did not recognize recovery—at least it was an insignificant factor. But the fundamental question in this respect is for how long a time we must make payments to the insured. As these cease at recovery as well as at death, we are really interested in the rate of cessation of payments instead of the death rate, and I believe that experience should be so analyzed—that is, that the combined rate of death and recovery should be compared with the expected deaths computed from Hunter's Table.

Consideration of the above may lead, however, to a false impression of our experience. Mr. Craig states that a large part of the financial loss of companies due to disability "is occasioned by setting up reserves, part of which may never be actually needed on account of recoveries." This follows from consideration of the above standpoint and it may be actually true, but we should not forget that most disability experience obtained, including the fundamental values of the reserves, is based upon Hunter's rate of mortality among disabled lives, which for the sake of convenience was taken as an average rate which produced proper premiums for the premium waiver benefit. This average rate, however, was obtained from an analyzed table in which the death rate during the first policy year following the year of disability varied from 3 1/2 times the average at age 30 to 3 times the average at age 50 and graded down to twice the average at age 59 where there is

332 Thirty-Fifth Annual Meeting

very little exposure. In other words, a ratio of actual to expected claims of about 300% should be expected during the first year following if the expected are calculated upon what is generally known as Hunter's Table. An experience of 300% of the expected during the first year following disability may in the long run average out to Hunter's standard and not, therefore, mean a less costly experience as would at first thought be supposed. The situation is really more critical than this because the death rate decreases so rapidly for several years after disability and yet the first year death rate used by Mr. Hunter really commenced at the policy anniversary following disability instead of at disability. Of course, it is probable that persons approved for disability now are not as totally disabled as were those whose experience entered into Hunter's Table, so that a lower death rate should now be expected, but it would appear wiser to calculate the ratio of actual to expected claims on the basis of the analyzed Select Table on which Hunter's Aggregate Table was based.

Our experience on claims approved in 1923 is remarkably similar to that given in Mr. Craig's first table as is evident from the following comparison. It may be further noted that our claims at high ages are increasing much more rapidly comparatively than at young ages.

| Metropolitan Disability % of Hunter's | | | The Travelers Disability % of Hunter's | | |
|--|--------|-------|---|--------|-------|
| Ages | Rate | Table | Ages | Rate | Table |
| 15-19 | .00110 | 215% | 15-20 | .00096 | 188% |
| 20-24 | .00109 | 200 | 21-25 | .00121 | 233 |
| 25-29 | .00107 | 199 | 26-30 | .00111 | 203 |
| 30-34 | .00100 | 172 | 31-35 | .00080 | 134 |
| 35-39 | .00100 | 142 | 36-40 | .00090 | 122 |
| 40-44 | .00121 | 128 | 41-45 | .00092 | 91 |
| 45-49 | .00154 | 116 | 46-50 | .00101 | 70 |
| 50-54 | .00203 | 100 | 51-55 | .00254 | 114 |
| 55-59 | .00351 | 100 | 56-59 | .00358 | 89 |

Mr. Craig gives an analysis of 1923 claims according to effective date of disability. Our experience is in a slightly different form, being according to the number of months claims are dated back as follows and being on the basis of in-

Discussion—Disability Benefit 333

dividual lives instead of policies. Among these claims were at least one in each policy year from the 1st to the 16th.

| No. Mos. Dated Back | No. of Individual Claims | No. Mos. Dated Back | No. of Individual Claims | No. Mos. Dated Back | No. of Individual Claims |
|---------------------------|--------------------------------|---------------------------|--------------------------------|---------------------------|--------------------------------|
| 1 | 10 | 14 | 5 | 27 | 4 |
| 2 | 8 | 15 | 8 | 28 | 2 |
| 3 | 23 | 16 | 2 | 30 | 1 |
| 4 | 18 | 17 | 4 | 32 | 1 |
| 5 | 18 | 18 | 1 | 33 | 1 |
| 6 | 22 | 19 | 3 | 34 | 2 |
| 7 | 20 | 21 | 4 | 36 | 1 |
| 8 | 7 | 22 | 2 | 38 | 1 |
| 9 | 15 | 23 | 1 | 43 | 2 |
| 10 | 9 | 24 | 6 | 44 | 2 |
| 11 | 7 | 25 | 2 | 53 | 1 |
| 12 | 6 | 26 | 2 | 54 | 1 |
| 13 | 6 | | | 88 | 1 |
| Average | | | | | |
| | | | | | 11.2 |

Analysis of our claims according to cause of disability as compared with the Metropolitan's is as follows:

| Cause of Disability | Number of Cases | |
|--|-----------------|---------------|
| | Metropolitan | The Travelers |
| Tuberculosis (Pulmonary) | 346 | 401 |
| Tuberculosis (other varieties) | 9 | 18 |
| Cancer | 16 | 54 |
| Acute Articular Rheumatism | 6 | |
| Anaemia | 7 | |
| Other general diseases | 5 | |
| Inflammation of the Brain | 9 | 31 |
| Locomotor Ataxia | 6 | 9 |
| Diseases of Spinal Cord | 11 | 42 |
| Apoplexy | 5 | 47 |
| Paralysis | 43 | 101 |
| Insanity | 106 | 125 |
| Other diseases of Nervous System | 16 | 29 |
| Blindness | 10 | 20 |
| Organic diseases of Heart | 11 | 32 |
| Diseases of the Arteries | — | 18 |
| Diseases of the Respiratory System | — | 10 |
| Diseases of the Digestive System | — | 19 |
| Diseases of the Genito-Urinary System | — | 28 |
| Diseases of the Skin and Cellular Tissue | — | 4 |
| Diseases of Bone & Organs of Locomotion | 8 | 15 |
| Traumatism | 16 | 21 |
| Fractures | 22 | 10 |
| External violence | 5 | 2 |
| Miscellaneous (less than 5 in any cause) | 38 | |
| Cause of Disability not specified | — | 4 |
| | 695 | 1,109 |

Mr. Craig raises the question as to whether Tuberculosis claims really constitute such a large proportion of total permanent disability claims or whether it is such a large proportion because the other claims have not yet developed. I am inclined to believe that it is at least partly due to the latter cause. I believe that our approval of Tuberculosis claims is as liberal as in the case of other experiences which have been published, but our experience is appreciably different from other experience because we have had policies in force with the disability provision since 1904. As is evident from the experience given above, we are incurring comparatively many more claims on policies that have been in force a number of years where the insured has reached the higher age and where more claims due to degenerative diseases are to be expected. In fact it is to be expected that the proportion of these causes will be much greater in the long run in our experience because such a large proportion of our business is recent that our exposure to Tuberculosis has been much greater comparatively than it has been to diseases that would develop several years after issuance of the policies. I think that our experience proves that other companies are not incurring the proportion of disability claims from these causes that they eventually will.

Dr. Ward—Dr. Root, have you a word for us?

Dr. Root—The disability feature added to life contracts is bound in itself to complicate the whole business of medical selection to a very considerable degree, and Mr. Craig has illustrated that and it has been referred to in the discussion. Two points are worthy of consideration—first, whether we are not almost bound to meet a form of selection against us in risks who are desirous of obtaining benefits prior to their death. I find the greatest difficulty in explaining to our agents the difference between a disability contract and a life contract, and they can never quite understand why we will issue life contracts when we refuse to issue disability. To my mind the difference is radical. In one you are insuring against

death, which everyone dreads. In the other you are insuring against disability which a large portion of people will tolerate if they are paid for it, and it seems to me the vital point we would make is that we have some idea of a man's fixed income, plus what he earns. I have seen it too often in compensation insurance, where an individual approaching middle life, who has been industrious, saving, and has acquired a few thousand or more in Liberty Bonds or some permanent investment, gets a little tired of his job, wakes up tired in the morning, and he goes to the doctor and the doctor says—"What you want is a winter in Florida. I'll write you a certificate for nervous prostration." He goes to Florida, and we pay the disability. Now, if we knew that man's income, what he sacrificed by giving up his job, we could add that to what he wants for the disability contract, and we could arrive at a safe conclusion as to what you could insure him for. Otherwise it will never be on a safe basis.

Another point is that in granting disability insurance on life contracts there are certain features of the history which should guide us very materially in acting. Our custom is something like this:

History of substandard or cases rated as substandard, of course we issue no permanent or total disability at all.

History of tuberculosis, either in the family recently or personal tuberculosis in the past, we refuse.

Goitre, for all ratings, we refuse.

Question of habits—we are very slow indeed in granting it.

Heart murmurs—we refuse.

Hypertrophy of the heart—we refuse.

Overweights—we grant.

Pleurisy—we refuse.

Any impairment of the pulse that is suspicious, we usually refuse.

Renal Colic—we refuse.

336 Thirty-Fifth Annual Meeting

Rheumatism—we absolutely refuse, whether acute, subacute, chronic or any of the ordinary arthritic types.

Syphilis—we refuse.

Ulcers—we refuse for all ratings.

The logic of that is obvious. In any one of those conditions they may be insured at standard or substandard rates and live forever, but they won't live and be in full earning capacity, especially if they have a contract by which you pay them for it.

Dr. Ward—Dr. Snow will discuss this paper.

Dr. Snow—Mr. Craig kindly sent me a copy of his paper a few days ago, so I have had an opportunity to study it, to appreciate its value and the amount of research entailed in its preparation. I have taken much comfort from the concluding words of his first paragraph—"Brief experience and may be regarded as still in the experimental stage."

To those of us whose Companies have incorporated the disability provisions in their Contracts the subject is fascinating not only as to selection but in the disposal of claims. We entered this field in 1914 and we entered with a handicap, as, with the exception of certain overweight groups, we limited acceptance of insurance risks to those considered standard by us. We were disposed to consider a standard insurance risk a standard disability risk except those with eye defects, mutilation, certain occupational hazards, etc. We even looked askance upon partial deafness. It was four years before we began to find ourselves. Of course we encountered the obvious, saw the increasing liberality of interpretation under court decisions, recognized the element of competition, and realized that, barring fraud, liberal interpretation was necessary.

Passing for the present, discussion of the Actuarial problems involved our figures began to show an unfavorable trend, chargeable largely to Pulmonary Tuberculosis and diseases of the nervous system. To make material betterment, we felt we must give primary attention to these outstanding sources

Discussion—Disability Benefit 337

of claims. Analysis of the nervous disease claims shows nothing but the axiomatic-syphilis must have been present in many of them, and in the entire group we find only four cases with any significant personal history and this remote. Under present conditions I do not think any of us expect to eliminate more than a small percentage of syphilitics to which cause a large part of the nervous disease disabilities are due, but as to Tuberculosis the situation seems more hopeful, and with this in mind we withdrew in 1921 both the waiver of premium and annuity provisions from all new contracts under age 21 and began a much closer selection under age 31 in the underweight groups. Whether or not the selection since that time can be considered satisfactory remains to be proven but the current figures indicate that it was at least a step in the right direction.

| Age | Percentage of Actual to be Expected |
|-------|-------------------------------------|
| 14-19 | 115.9 |
| 20-24 | 143.8 |
| 25-29 | 198.2 |
| 30-34 | 126.4 |
| 35-39 | 68.9 |
| 40-44 | 80.3 |
| 45-49 | 87.5 |
| 50-54 | 41.8 |
| 55-59 | 50.9 |
| Total | 89.7 |

These figures in trend agree substantially with those shown by Mr. Craig. It should be noted that the 14-19 experience is very small, disability not having been issued at those ages since 1921, but the group 20-34 is significant, and an analysis of the claims shows the result more strikingly. The three groups show an aggregate of 185 claims, 37% of all claims approved during that period, and of this 185 150 were under weights, 35 of the cases standard weight or better though only 11 of the 35 were 10 lbs. or more over standard and we believe that most of the other 24 were estimate weights rather than actual weights. In the 5-year group, 31-35, there were 25 approved claims on overweights and only 2 where the

338 Thirty-Fifth Annual Meeting

weight was more than 5 lbs. over standard. The early termination of these claims is shown by the following table:

| To Date June 1924 | |
|---------------------------------------|-----|
| Tuberculosis | |
| Total claims | 244 |
| Total claims still existing | 111 |
| Total claims terminated | 133 |
| Claims terminated under 1 year | 70 |
| Claims terminated under 2 years | 38 |
| Claims terminated under 3 years | 18 |
| Claims terminated under 4 years | 4 |
| Claims terminated under 5 years | 2 |
| Claims terminated under 6 years | 1 |
| | 133 |
| Total claims ending in death | 74 |
| Total claims ending in recovery | 59 |
| Total claims existing | 111 |
| | 244 |
| Nervous Diseases | |
| Total claims | 93 |
| Total claims existing | 51 |
| Total claims terminated | 42 |
| Total claims terminated under 1 year | 19 |
| Total claims terminated under 2 years | 14 |
| Total claims terminated under 3 years | 5 |
| Total claims terminated under 4 years | 3 |
| Total claims terminated under 7 years | 1 |
| | 42 |
| Total claims ending in death | 33 |
| Total claims ending in recovery | 9 |
| Total claims existing | 51 |
| | 93 |

46% of all tuberculosis claims have already terminated and 54% still exist. In the entire group of 244 consecutive claims, however, only 7 showed one case of Tuberculosis in the immediate family. Under nervous diseases 55% exist, 45% terminated already. Total experience all causes to correct date, in force 287, 49.8%: died 185, 32.1%: recovered 100, 19.8%.

In connection with this study of underweights it must be

noted that the result is a composite of the entire selection made up of applicants qualifying under Life acceptance rules prior to 1920 and those later selected by the elimination of entrants under 21 and a much more rigid weight selection thereafter.

It is apparent that the degree of selection will materially affect the result both as to incidence of disability and the recovery and death rate among the disabled; that the class of business will have its effect, because among people in well to do circumstances fewer claims will be made even when claims arise; that the average age of the business affects the rate and is shown by practically all observation. It is also observable that a variance in figures would result from immediate approval or after any specific time. It is our belief that Hunter's table is not representative in incidence of claims. The very data on which the table is based must lead to this conclusion aside from actual experience.

On account of the brief period of observation our experience is necessarily limited to early claims. In connection with later claims two problems arise, first, unmarried women after marriage when the necessity of following a gainful occupation ceases and where the exact physical status is difficult to prove. We now attach a rider to all Policies on all female lives. "If the insured is a female, the foregoing provisions as to permanent total disability shall cease to be in force upon marriage and the premium therefor shall be no longer payable." Second, those approaching 60 or 65, who for any reason no longer wish to pursue an occupation for gain and whose physical status though indeterminate may nevertheless give rise to claims which cannot be denied. Apparently we must treat these as occasion demands. Toward the conclusion of Mr. Craig's paper he mentions five questions to be covered on application. We endeavor to cover them all except the third which we hope to determine through inspection and a question covering total existing disability coverage.

340 Thirty-Fifth Annual Meeting

Focal infection as stated may be a source of later trouble but personally I should have more fear of the undiscovered and untreated infection than that already recognized and treated.

Dr. Porter—Mr. President—On behalf of the Association I move that we extend to the Mutual Benefit Life Insurance Company, its officers and its medical corps, our heartfelt thanks for the delightful courtesy which they have extended to us during this meeting. Everything has been done for the comfort of the members, and for the scientific as well as the social success of the meeting.

Dr. Beckett seconded the motion and it was carried.

Dr. Ward introduced the incoming President, Dr. C. F. S. Whitney, who in a few words expressed his appreciation of the honor paid him in electing him as President and also of the compliment thus extended to the representatives of the smaller companies.

On motion the meeting adjourned *sine die*.

The Annual Dinner of the Association was held at the Robert Treat Hotel, Newark, N. J., on the evening of Thursday, October 23rd, 1924. The following members and guests were present:

| | | |
|-----------------|---------------------|-----------------------|
| Adams, John L. | Bradshaw, W. M. | Daley, R. M. |
| Allen, E. H. | Brown, C. T. | Dewis, E. G. |
| Anderson, H. B. | Byrd, Dr. | Dillard, H. K. |
| Aten, W. B. | | Dingman, W. H. |
| Bailey, W. C. | Chapin, F. W. | Duffield, E. D. (Mr.) |
| Baker, H. A. | Christiernin, C. L. | Dwight, E. W. |
| Bartlett, W. B. | Clark, C. P. | |
| Battle, T. J. | Colton, E. A. | |
| Beckett, W. W. | Cragin, D. B. | Eakins, O. M. |
| Bennett, C. D. | Crawford, G. E. | Exton, W. G. |
| Birchard, C. C. | | |

Annual Dinner**341**

| | | |
|-----------------------|----------------------|---------------------|
| Finnerud, Dr. | Lair, M. M. | Schadt, Geo. L. |
| Fisk, Lyman | Lamb, W. P. | Scholz, S. B. Jr. |
| Fitzgerald, Paul | | Shaw, G. H. |
| Fraser, R. A. | | Smith, M. K. |
| Gadd, S. W. | MacKenzie, L. F. | Smith, T. A. |
| Ganot, F. I. | Means, S. W. | Snow, M. |
| Gore, John K. (Mr.) | Medd, John C. | Speer, H. B. |
| Griswold, A. H. | McCradden, F. H. | Stanton, S. C. |
| Grosvenor, F. L. | McMahon, T. F. | Sykes, Lawrence |
| Hagney, F. W. | Miller, Jas. Alex. | |
| Hall, G. W. C. | Milroy, W. F. | |
| Hall, J. B. | Olsen, M. I. | Tiemann, P. E. |
| Hardin, John L. (Mr.) | | Thornton, W. E. |
| Härnden, F. | Papps, Percy (Mr.) | Truitt, F. L. |
| Hobbs, A. B. | Patton, J. A. | Turner, J. P. |
| Hunter, Arthur (Mr.) | Pauli, W. O. | |
| Huston, Ross | Peterson, W. A. | Van Dervoort, C. A. |
| Hutchinson, W. G. | Piper, C. B. | Van Kleeck, E. |
| Hutton, Lefferts | Phelps, J. S. | Van Wagenen, G. A. |
| Irwin, C. B. | Pollard, J. E. | |
| Jaquith, W. A. | Porter, W. E. | Ward, William R. |
| Jenney, F. L. B. | Priestley, J. T. | Watson, W. P. |
| Johann, A. E. | | Weisse, F. S. |
| Kanouse, G. E. | Reiter, W. A. | Wehner, W. H. |
| Kingsbury, F. S. | Rhodes, Edward (Mr.) | Wells, F. L. |
| Kinney, J. E. | Rockwell, T. H. | Whicher, C. M. |
| Kissock, R. J. | Rolph, F. W. | Whitney, C. F. S. |
| Knight, A. S. | Root, E. K. | Willard, T. H. |
| Kyle, E. B. | Ross, A. R. | Willis, R. L. |
| | Rowley, R. L. | Wilson, M. C. |
| | Russell, E. F. | Wood, Glenn |
| | | Young, H. H. |

REMINISCENCES AND AFTER DINNER TALK

DR. GEORGE A. VAN WAGNER

Oct. 23, 1924 at Annual Dinner of the Medical Directors' Association.

Fellow Medical Directors:

I may very appropriately greet you in the words of the Roman Gladiators as they appeared in the arena before Caesar to fight for their lives—"We who are about to die salute you," for they tell me, and the records bear them out, that I am the last of the 28 "good fellows" who attended the preliminary meeting of this Association, 35 years ago. I find I am the only one living of the 18 who, six months later, attended the first meeting of our Society, and adopted the constitution and by-laws under which it began its wonderful mission of scientific and social beneficence; and that I am even the only one left of the 34 whose names were on its first roll call.

This is discouraging enough, but when I turn to our friends, the actuaries, to ask what my present expectancy of life is, they quote their well-known rule—"2/3 of the difference between your age and 80"—Gentlemen, I ask you, "What comfort is there in an equation of that sort, for a fellow who has already celebrated his 79th birthday?" These actuaries are canny fellows; they promise me, when young, to assure or insure my life for a certain yearly payment; but when I read "the bond" they gave me, *it* said "payable personally, IF you reach the age of 96" (we life insurance men know that 96 is the age at which "the insured is mathematically dead, even if he is alive, and can eat, drink, and make merry," as we do tonight). However, they added by way of comfort, "If you don't happen to reach this good old age, our company will pay the money to your heirs, making them happier than if you had lived so long."

Now for some recollections of the early days of our Association, which I have been asked to give. Please keep in mind the fact that previous to 1889 we Medical Directors had no personal acquaintance. Necessary correspondence was limited, and as formal as that in which a military man addresses his superior officer, or a Government official replies to a private citizen. Replies were as short as possible, and none too explicit; for competition was so bitter that several Companies refused all communication with certain others. There was a state of business war. It was then that Dr. John M. Keating of the Penn Mutual invited the Medical Directors of a number of Companies to meet at the Union League Club of New York, on the evening of May 29th, 1889, to become acquainted—please notice that the home office of the Penn. Mutual is at Philadelphia, which is beautiful Greek for brotherly love; and that this meeting was to be held at the Union League Club—a most appropriate place. Under such favorable omens our Association was conceived, and later born.

Twenty-eight responded to this invitation; Dr. Keating made a short address reciting the enormous volume and importance of Life Insurance; our responsibility in selection of applicants, and referring to the fact that we were unacquainted with the Medical Men we corresponded with, he introduced us. The evening passed so delightfully it was proposed to repeat it; and finally a resolution was unanimously passed, and a Committee appointed to draft a Constitution and by-laws for a permanent organization. Six months later our Constitution was adopted; and, at this *very first* meeting, scientific work was begun by an able paper from Dr. Bernacki of the Germania Life Co.—“The effect on longevity of drinking large quantities of beer” (this was 35 years ago). Our present day prohibitionists would be charmed with the damaging statistics from Prof. Oertel’s clinic at Munich, the home of “the best beer in this world”; but the anti’s would be delighted

344 Thirty-Fifth Annual Meeting

with his conclusion, that the damage was due to the quantity of pure water, and not to the alcohol. The acquaintances made at these early meetings had an immediate effect on the character of our medical correspondence, which became personal, and contained courteous and complete answers to all questions asked, all of which proves that more can be done by "Mutual Aid" than by Mutual Competition.

At the second regular meeting an able paper on that perennial and *yet* unsettled subject, "Albuminurea in the apparently healthy," was read by Dr. Wm. B. Davis of the Union Central. From these modest beginnings, our scientific work has developed, until we now have ample and reliable data for the selection of risks.

The first combined effort in statistics was fathered by Dr. Shepherd of the Conn. Mutual in 1905, resulting in our table of height and weight at different ages in 1907—a monument to Dr. Shepherd. The second combined statistics were those of the medical directors' "Specialized Mortality investigation" in 1908. When it was proposed to extend this valuable work, the actuarial society, organized the same year as our own, joined the Med. directors in producing the "Combined Medico-Actuarial Mortality investigation," begun in 1909, and published in 1912—1914—a stupendous, and magnificent work which has given us five marvelous volumes of tables based on *actual facts* deduced from the mortality of some two million insured lives, contributed by forty-three companies. This work is a lasting monument to the combined Medical Directors and actuaries who composed the committee in charge, and especially to Mr. Hunter of the N. Y. Life, who devoted his time and labor to this work so lavishly.

Forty-six years ago (ten years before the birth of this association) when I became a medical director of our company, it was impossible to find any works on Life Insurance

"Selection"; consequently every company selected its risks, according to the varied experience and views of its own Medical Board. It is *notorious* that doctors, lawyers and ministers always disagree; so that what one company declined, others as good could always be found anxious to accept. This fault was recognized early; and at our 6th meeting, Dr. Stebbins of the Mass. Mutual, read a scholarly paper on "Uniformity in Life Insurance," claiming especially that under like circumstances what was good for one must be good for all others.

The first member to join our Association by election was our friend, Fisher, of the North Western. His election was one of the best things the Association ever did for itself, and you all know what he had done for it.

When our society was 16 years old, it had eighty-three members, representing forty-one companies; and exchange of medical information became so frequent that a code was invented by Dr. Rogers to simplify and limit correspondence. This M. I. B., as he has gradually developed it, is of inestimable advantage to us, to our companies, and even to the insured.

In closing, I will recall a very few of our earliest members. Dr. Russell, of the Aetna, was the oldest and most conservative; his remark, "When in doubt, I always allow a more ambitious company to take the case," was characteristic.

Frank Wells, of the John Hancock, one of our first presidents, was our "exquisite little gentleman," slight in figure, faultlessly dressed, he was as erect and quick as a West Point cadet. While Dr. Tabb, of the Virginia Life, was the portly and genial representative of the F. F. Vs., a typical "Southern Gentleman."

Edgar Holden, my senior on the Board of the Mutual Benefit, a charming man personally, was a fine scholar and very able writer.

346 Thirty-Fifth Annual Meeting

George Winston, of the Mutual Life, was permanent chairman of our dinner committee; an able disciple of the great French Epicure Savarin; he knew "everything good to eat and drink," and all that makes the difference between "feeding" and "dining." He was most ably followed by our friend, Porter. Edward Curtis, of the Equitable, will not be forgotten by those fortunate enough to hear his after-dinner story of "G. Washington and his little hatchet," as he rendered it in song.

Nor can we forget the wonderfully impressive way in which Dr. Hammil of the Prudential, repeated the poem, "An Apostrophy to a Skull."

I can simply recall the names of Lambert of the Equitable, Gillette of the Mutual Life, Huntington of the New York Life, McKnight of the North Western, Marsh of the Mutual Life, Gage of the State Mutual, Foster of the Union Central, Paddock of the Berkshire, Rex of the Penn. Mutual, etc. Experience, our profession, and especially our Life Insurance work remind us our own names will soon be added to this list. "Death" is but *one* of the adventures of "Life." Its especial importance lies simply in the fact that it is the last—as far as we know. Life is the factor of real importance; and in closing, let me quote the last few lines of Bryant's magnificent poem—*Thanatopsis*.

"So live that when thy summons comes to join
The innumerable caravan that moves
To that mysterious realm where
Each shall take his chamber in the silent halls of death,
Thou go, not like the galley slave at night,
Scourged to his dungeon: but sustained and soothed
By an unfaltering trust, approach thy grave
Like one that draws the drapery of his couch
About him, and lies down to pleasant dreams."

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE
INSURANCE MEDICAL DIRECTORS.

| | |
|------------------------------|--|
| Frederick R. Abbe, M. D. | Columbian National, Bos- ton, Mass. |
| John L. Adams, M. D. | Metropolitan, New York, N. Y. |
| Edwin H. Allen, M. D. | John Hancock, Boston, Mass. |
| Charles D. Alton, M. D. | Connecticut Mutual, Hartford, Conn. |
| John W. Amesse, M. D. | Capitol Life of Colorado, Denver, Colo. |
| Harry B. Anderson, M. D. | Imperial Life, Toronto, Ont. |
| Thomas D. Archibald, M. D. | North American, Toron- to, Ont. |
| Edward McP. Armstrong, M. D. | Mutual, New York, N. Y. |
| William Armstrong, M. D. | Connecticut General, Hartford, Conn. |
| William B. Aten, M. D. | Security Mutual, Bing- hamton, N. Y. |
| Malcolm O. Austin, M. D. | West Coast, San Fran- cisco, Calif. |
| Henry A. Baker, M. D. | Kansas City Life, Kansas City, Mo. |
| G. Holbrook Barber, M. D. | Manhattan, New York, N. Y. |

348 Thirty-Fifth Annual Meeting

| | |
|--------------------------------|---|
| William B. Bartlett, M. D. | John Hancock, Boston, Mass. |
| John T. J. Battle, M. D. | Jefferson Standard, Greensboro, N. C. |
| Wesley W. Beckett, M. D. | Pacific Mutual, Los Angeles, Calif. |
| Charles D. Bennett, M. D. | Mutual Benefit, Newark, N. J. |
| Thomas W. Bickerton, M. D. | New York Life, New York, N. Y. |
| Edward B. Bigelow, M. D. | State Mutual, Worcester, Mass. |
| Albert W. Billing, M. D. | Equitable, New York, N. Y. |
| Cecil C. Birchard, M. D. | Sun Life, Montreal, Quebec. |
| Arthur B. Bisbee, M. D. | National, Montpelier, Vt. |
| David N. Blakely, M. D. | New England Mutual, Boston, Mass. |
| Robert J. Blanchard, M. D. | Great West, Winnipeg, Manitoba. |
| William M. Bradshaw, M. D. | Mutual, New York, N. Y. |
| Frederick G. Brathwaite, M. D. | Equitable, New York, N. Y. |
| Chester T. Brown, M. D. | Prudential, Newark, N. J. |
| William H. Browne, M. D. | American Life, Detroit, Mich. |
| Thomas W. Burrows, M. D. | Central Life, Ottawa, Ill. |
| Joseph T. Cabaniss, M. D. | Travelers, Hartford, Conn. |
| Frank H. Carber, M. D. | Mutual, New York, N. Y. |
| Frank W. Chapin, M. D. | Home Life, New York, N. Y. |
| Laurence D. Chapin, M. D. | Massachusetts Mutual, Springfield, Mass. |

List of Members

349

| | |
|--------------------------------|---|
| John P. Chapman, M. D. | Penn. Mutual, Philadelphia, Pa. |
| Charles L. Christiernin, M. D. | Metropolitan, New York, N. Y. |
| Charles P. Clark, M. D. | Mutual Benefit, Newark, N. J. |
| Henry Colt, M. D. | Berkshire, Pittsfield, Mass. |
| Edwin A. Colton, M. D. | National, Montpelier, Vt. |
| Henry W. Cook, M. D. | Northwestern National, Minneapolis, Minn. |
| John N. Coolidge, M. D. | Metropolitan, New York, N. Y. |
| Robert M. Daley, M. D. | Equitable, New York, N. Y. |
| Edwin Grafign Dewis, M. D. | Prudential, Newark, N. J. |
| Wilton E. Dickerman, M. D. | Aetna, Hartford, Conn. |
| Henry K. Dillard, M. D. | Penn. Mutual, Philadelphia, Pa. |
| William W. Dinsmore, M. D. | Travelers, Hartford, Conn. |
| Percy G. Drake, M. D. | Travelers, Hartford, Conn. |
| Edwin W. Dwight, M. D. | New England Mutual, Boston, Mass. |
| O. M. Eakins, M. D. | Reliance, Pittsburgh, Pa. |
| Zenas H. Ellis, M. D. | Connecticut General, Hartford, Conn. |
| Calvin H. English, M. D. | Lincoln National, Ft. Wayne, Ind. |
| William G. Exton, M. D. | Prudential, Newark, N. J. |
| John Ferguson, M. D. | Excelsior, Toronto, Ont. |

350 Thirty-Fifth Annual Meeting

| | |
|----------------------------|---|
| John W. Fisher, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| Paul Fitzgerald, M. D. | Prudential, Newark, N. J. |
| Robert A. Fraser, M. D. | New York Life, New York, N. Y. |
| Dudley Fulton, M. D. | Occidental Life, Los An- geles, Calif. |
| Samuel W. Gadd, M. D. | Philadelphia Life, Phila- delphia, Pa. |
| Homer Gage, M. D. | State Mutual, Worcester, Mass. |
| Frank Irving Ganot, M. D. | Prudential, Newark, N. J. |
| William S. Gardner, M. D. | Mutual Life, New York, N. Y. |
| Donald M. Gedge, M. D. | Metropolitan, New York, N. Y. |
| Arthur Geiringer, M. D. | Equitable, New York, N. Y. |
| Alvah H. Gordon, M. D. | London & Scottish, Mon- treal, Que. |
| Angus Graham, M. D. | London Life, London, Ont. |
| LeRoy C. Grau, M. D. | Travelers, Hartford, Conn. |
| Arthur H. Griswold, M. D. | Phoenix Mutual, Hart- ford, Conn. |
| Frank L. Grosvenor, M. D. | Travelers, Hartford, Conn. |
| George C. Hall, M. D. | Life Ins. Co. of Virginia, Richmond, Va. |
| Joseph B. Hall, M. D. | Connecticut Mutual, Hartford, Conn. |
| William F. Hamilton, M. D. | Sun Life, Montreal, Que. |

List of Members

351

| | |
|------------------------------|---------------------------------------|
| William J. Hammer, M. D. | New York Life, New York, N. Y. |
| George A. Harlow, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| Whitefield Harral, M. D. | Southwestern Life, Dallas, Tex. |
| Calvin L. Harrison, M. D. | New York Life, New York, N. Y. |
| Ernest M. Henderson, M. D. | Confederation Life, Toronto, Ont. |
| Charles R. Henry, M. D. | State Life, Indianapolis, Ind. |
| John U. Hobach, M. D. | Penn Mutual, Philadelphia, Pa. |
| Angier B. Hobbs, M. D. | New York Life, New York, N. Y. |
| Wm. W. Hobson, M. D. | Reliance Life, Pittsburgh, Pa. |
| Eugene M. Holden, M. D. | Metropolitan, New York, N. Y. |
| Jerome F. Honsberger, M. D. | Mutual Life of Canada, Waterloo, Ont. |
| Ross Huston, M. D. | Bankers, Des Moines, Iowa. |
| James P. Hutchinson, M. D. | Penn Mutual, Philadelphia, Pa. |
| William G. Hutchinson, M. D. | Michigan Mutual, Detroit, Mich. |
| Henry S. Hutchinson, M. D. | Canada Life, Toronto, Ont. |
| Lefferths Hutton, M. D. | Mutual Life, New York, N. Y. |
| Phineas H. Ingalls, M. D. | Aetna Life, Hartford, Conn. |

352 Thirty-Fifth Annual Meeting

| | |
|-----------------------------|---|
| Charles B. Irwin, M. D. | North American, Chicago, Ill. |
| Walter A. Jaquith, M. D. | National, Chicago, Ill. |
| Benjamin Y. Jaudon, M. D. | Missouri State Life, St. Louis, Mo. |
| Frank L. B. Jenney, M. D. | Federal Life, Chicago, Ill. |
| Albert O. Jimenis, M. D. | Metropolitan, New York, N. Y. |
| Albert E. Johann, M. D. | Bankers, Des Moines, Iowa. |
| George E. Kanouse, M. D. | Prudential, Newark, N. J. |
| Edward B. Kellogg, M. D. | John Hancock, Boston, Mass. |
| Morris L. King, M. D. | New York Life, New York, N. Y. |
| Robert J. Kissock, M. D. | Metropolitan, New York, N. Y. |
| Augustus S. Knight, M. D. | Metropolitan, New York, N. Y. |
| William P. Lamb, M. D. | Prudential, Newark, N. J. |
| John L. Larway, M. D. | State Life of Indiana, Indianapolis, Ind. |
| Ernest H. Lines, M. D. | New York Life, New York, N. Y. |
| John Mason Little, M. D. | New England Mutual, Boston, Mass. |
| John M. Livingston, M. D. | Mutual Life of Canada, Waterloo, Ont. |
| Robert L. Lounsherry, M. D. | Security Mutual, Binghamton, N. Y. |
| Lewis F. MacKenzie, M. D. | Prudential, Newark, N. J. |

List of Members

353

| | |
|-------------------------------|-------------------------------------|
| Charles N. McCloud, M. D. | Minnesota Mutual, St. Paul, Minn. |
| Charleton B. McCulloch, M. D. | State Life, Indianapolis, Ind. |
| Thomas F. McMahon, M. D. | Manufacturers, Toronto, Ont. |
| Charles Maertz, M. D. | Union Central, Cincinnati, Ohio. |
| Robert W. Mann, M. D. | Imperial, Toronto, Ont. |
| William S. Manners, M. D. | Metropolitan, New York, N. Y. |
| Henry A. Martelle, M. D. | Connecticut Mutual, Hartford, Conn. |
| Charles F. Martin, M. D. | Standard Life, Montreal, Que. |
| Oscar F. Maxon, M. D. | Franklin Life, Springfield, Ill. |
| * | |
| Paul Mazzuri, M. D. | New York Life, New York, N. Y. |
| Samuel W. Means, M. D. | Metropolitan, New York, N. Y. |
| John C. Medd, M. D. | Metropolitan, New York, N. Y. |
| George L. Megargee, M. D. | Metropolitan, New York, N. Y. |
| Archibald Mercer, M. D. | Mutual Benefit, Newark, N. J. |
| William F. Milroy, M. D. | Bankers Reserve, Omaha, Neb. |
| James T. Montgomery, M. D. | Southland, Dallas, Texas. |
| William D. Morgan, M. D. | Phoenix Mutual, Hartford, Conn. |
| William Muhlberg, M. D. | Union Central, Cincinnati, Ohio. |

354 Thirty-Fifth Annual Meeting

| | |
|------------------------------|--|
| John P. Munn, M. D. | United States, New York, N. Y. |
| James H. North, M. D. | New York Life, New York, N. Y. |
| Edwin M. Northcott, M. D. | Union Mutual, Portland, Me. |
| Ralph B. Ober, M. D. | Massachusetts Mutual, Springfield, Mass. |
| Jay Bergen Ogden, M. D. | Metropolitan, New York, N. Y. |
| Herbert Old, M. D. | Provident Life & Trust, Philadelphia, Pa. |
| Martin I. Olsen, M. D. | Central Life, Des Moines, Iowa. |
| Brace W. Paddock, M. D. | Berkshire Life, Pittsfield, Mass. |
| Howard A. Pardee, M. D. | United States Life, New York, N. Y. |
| George W. Parker, M. D. | Peoria Life, Peoria, Ill. |
| J. Allen Patton, M. D. | Prudential, Newark, N. J. |
| William O. Pauli, M. D. | Union Central, Cincin- nati, Ohio. |
| Harry S. Pearse, M. D. | Equitable, New York, N. Y. |
| William A. Peterson, M. D. | Mutual Trust, Chicago, Ill. |
| John S. Phelps, M. D. | Columbian National, Bos- ton, Mass. |
| Charles B. Piper, M. D. | Guardian Life, New York, N. Y. |
| Joseph E. Pollard, M. D. | Prudential, Newark, N. J. |
| William Evelyn Porter, M. D. | Mutual, New York, N. Y. |
| Albert T. Post, M. D. | Equitable, New York, N. Y. |

List of Members

355

| | |
|------------------------------|---|
| James T. Priestley, M. D. | Royal Union Mutual, Des Moines, Iowa. |
| Walter A. Reiter, M. D. | Mutual Benefit, Newark, N. J. |
| Frank P. Righter, M. D. | Atlantic Life, Richmond, Va. |
| William B. Robbins, M. D. | New England Mutual, Boston, Mass. |
| Thomas H. Rockwell, M. D. | Equitable Life, New York, N. Y. |
| Oscar H. Rogers, M. D. | New York Life, New York, N. Y. |
| Fred W. Rolph, M. D. | Confederation, Toronto, Ont. |
| Edward K. Root, M. D. | Aetna, Hartford, Conn. |
| Robert L. Rowley, M. D. | Phoenix Mutual, Hartford, Conn. |
| Eugene F. Russell, M. D. | Mutual, New York, N. Y. |
| H. Crawford Scadding, M. D. | Canada Life, Toronto, Ont. |
| Samuel B. Scholz, Jr., M. D. | Massachusetts Mutual Springfield, Mass. |
| Henry H. Schroeder, M. D. | Mutual, New York, N. Y. |
| Arthur L. Sherrill, M. D. | Equitable, New York, N. Y. |
| Daniel M. Shewbrooks, M. D. | Lincoln National, Fort Wayne, Ind. |
| Donald W. Skeel, M. D. | Occidental, Los Angeles, Calif. |
| James M. Smith, M. D. | American Central, Indianapolis, Ind. |
| Malcolm K. Smith, M. D. | Prudential, Newark, N. J. |

356 Thirty-Fifth Annual Meeting

| | |
|-------------------------------|---|
| Morton Snow, M. D. | Massachusetts Mutual, Springfield, Mass. |
| Marion Souchon, M. D. | Pan-American, New Orleans, La. |
| Howard B. Speer, M. D. | Metropolitan, New York, N. Y. |
| Henry F. Starr, M. D. | Southern Life & Trust, Greensboro, N. C. |
| John B. Steele, M. D. | Volunteer State Life, Chattanooga, Tenn. |
| George S. Strathy, M. D. | Canada Life, Toronto, Ont. |
| Lawrence C. Sykes, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| William Thorndike, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| Walter E. Thornton, M. D. | Lincoln National, Fort Wayne, Ind. |
| Paul E. Tiemann, M. D. | New York Life, New York, N. Y. |
| Harry Toulmin, M. D. | Penn Mutual, Philadelphia, Pa. |
| Frank L. Truitt, M. D. | Reserve Loan, Indianapolis, Ind. |
| John S. Turner, M. D. | Southland, Dallas, Texas. |
| Joseph P. Turner, M. D. | Jefferson Standard, Greensboro, N. C. |
| Henry G. Tuttle, M. D. | Metropolitan, New York, N. Y. |
| Charles A. VanDervoort, M. D. | Fidelity Mutual, Philadelphia, Pa. |
| Euen VanKleeck, M. D. | Travelers, Hartford, Conn. |

List of Members

357

| | |
|------------------------------|--|
| George A. VanWagenen, M. D. | Mutual Benefit, Newark, N. J. |
| Albert A. Wagner, M. D. | Reliance Life, Pittsburgh, Pa. |
| Charles E. Waits, M. D. | Southern States, Atlanta, Ga. |
| William R. Ward, M. D. | Mutual Benefit, Newark, N. J. |
| William Perry Watson, M. D. | Prudential, Newark, N. J. |
| William E. H. Wehner, M. D. | Fidelity Mutual, Phila- delphia, Pa. |
| Faneuil S. Weisse, M. D. | Mutual, New York, N. Y. |
| Ernest A. Wells, M. D. | Aetna, Hartford, Conn. |
| Fred L. Wells, M. D. | Equitable of Iowa, Des Moines, Iowa. |
| David E. W. Wenstrand, M. D. | Northwestern Mutual, Milwaukee, Wis. |
| Charles D. Wheeler, M. D. | State Mutual, Wor- cester, Mass. |
| Chester F. S. Whitney, M. D. | Home Life, New York, N. Y. |
| Thomas H. Willard, M. D. | Metropolitan, New York, N. Y. |
| Richard Lee Willis, M. D. | Mutual, New York, N. Y. |
| Charles H. Willits, M. D. | Provident Mutual, Phila- delphia, Pa. |
| Gordon Wilson, M. D. | Maryland Life, Balti- more, Md. |
| McLeod C. Wilson, M. D. | Travelers, H a r t f o r d, Conn. |

358 Thirty-Fifth Annual Meeting

HONORARY MEMBERS

| | |
|---------------------------|------------------|
| John W. Brannan, M. D. | New York, N. Y. |
| John K. Gore | Newark, N. J. |
| Arthur Hunter | New York, N. Y. |
| Thomas Glover Lyon, M. D. | London, England. |
| Edward E. Rhodes | Newark, N. J. |
| Archibald A. Welch | Hartford, Conn. |
| Granville M. White, M. D. | New York, N. Y. |

COMPANIES AND THEIR REPRESENTATIVES

| | |
|--|---|
| Aetna Life, Hartford, Conn. | { W. E. Dickerman, M. D. P. H. Ingalls, M. D. E. K. Root, M. D. E. A. Wells, M. D. |
| American Central Life, Indianapolis, Ind. | J. M. Smith, M. D. |
| American Life, Detroit, Mich. | W. H. Browne, M. D. |
| Atlantic Life, Richmond, Va. | F. P. Righter, M. D. |
| Bankers Life, Des Moines, Iowa | { Ross Huston, M. D. Albert E. Johann, M. D. |
| Bankers Reserve Life, Omaha, Neb. | W. F. Milroy, M. D. |
| Berkshire Life, Pittsfield, Mass. | { Henry Colt, M. D. B. W. Paddock, M. D. |
| Canada Life Assurance Co. Toronto, Ont., Can. | { H. S. Hutchison, M. D. H. C. Scadding, M. D. G. S. Strathy, M. D. |
| Capitol Life of Colorado, Denver, Colo. | John W. Amesse, M. D. |
| Central Life of Des Moines, Des Moines, Iowa | M. I. Olsen, M. D. |
| Columbian National Life, Boston, Mass. | { F. R. Abbe, M. D. J. S. Phelps, M. D. |
| Confederation Life Ass'n, Toronto, Ont., Can. | { E. M. Henderson, M. D. F. W. Rolph, M. D. |

360 Thirty-Fifth Annual Meeting

| | |
|---|--|
| Connecticut General Life, Hartford, Conn. | { William Armstrong, M. D. Z. H. Ellis, M. D. |
| Connecticut Mutual Life, Hartford, Conn. | { C. D. Alton, M. D. J. B. Hall, M. D. H. A. Martelle, M. D. |
| Central Life of Illinois, Ottawa, Ill. | T. W. Burrows, M. D. |
| Equitable Life Assur. Soc., New York, N. Y. | { A. W. Billing, M. D. F. G. Brathwaite, M. D. R. M. Daley, M. D. Arthur Geiringer, M. D. H. S. Pearse, M. D. A. T. Post, M. D. T. H. Rockwell, M. D. A. H. Sherrill, M. D. |
| Equitable Life of Iowa, Des Moines, Iowa | F. L. Wells, M. D. |
| Excelsior Life, Toronto, Ont., Can. | John Ferguson, M. D. |
| Federal Life, Chicago, Ill. | F. L. B. Jenney, M. D. |
| Fidelity Mutual Life, Philadelphia, Pa. | { C. A. VanDervoort, M. D. W. H. E. Wehner, M. D. |
| Franklin Life, Springfield, Ill. | O. F. Maxon, M. D. |
| Great West Life Assur. Co., Winnipeg, Man., Can. | R. J. Blanchard, M. D. |
| Guardian Life, New York, N. Y. | C. B. Piper, M. D. |

Companies and Representatives 361

| | |
|--|--|
| Home Life, New York, N. Y. | { F. W. Chapin, M. D. C. F. S. Whitney, M. D. |
| Illinois Life, Chicago, Ill. | Glenn Wood, M. D. |
| Imperial Life Assur. Co., Toronto, Ont., Can. | { H. B. Anderson, M. D. R. W. Mann, M. D. |
| Jefferson Standard Life, Greensboro, N. C. | { J. T. J. Battle, M. D. J. P. Turner, M. D. |
| John Hancock Mutual Life, Boston, Mass. | { E. H. Allen, M. D. W. B. Bartlett, M. D. E. B. Kellogg, M. D. |
| Kansas City Life, Kansas City, Mo. | H. A. Baker, M. D. |
| Life Ins. Co. of Virginia, Richmond, Va. | G. C. Hall, M. D. |
| Lincoln National Life, Fort Wayne, Ind. | { C. H. English, M. D. D. M. Shewbrooks, M. D. W. E. Thornton, M. D. |
| London & Scot. Assur. Ass'n, Ltd., Montreal, Que., Can. | A. H. Gordon, M. D. |
| London Life, London, Ont., Can. | Angus Graham, M. D. |
| Manhattan Life, New York, N. Y. | G. H. Barber, M. D. |

362 Thirty-Fifth Annual Meeting

| | |
|--|---|
| Manufacturers Life, Toronto, Ont., Can. | T. F. McMahon, M. D. |
| Maryland Life, Baltimore, Md. | Gordon Wilson, M. D. |
| Massachusetts Mutual Life, Springfield, Mass. | L. D. Chapin, M. D. R. B. Ober, M. D. S. B. Scholz, Jr., M. D. Morton Snow, M. D. |
| Metropolitan Life, New York, N. Y. | J. L. Adams, M. D. C. L. Christiernin, M. D. J. N. Coolidge, M. D. D. M. Gedge, M. D. E. M. Holden, M. D. A. O. Jimenis, M. D. R. J. Kissock, M. D. A. S. Knight, M. D. W. S. Manners, M. D. S. W. Means, M. D. J. C. Medd, M. D. G. L. Megargee, M. D. J. B. Ogden, M. D. H. B. Speer, M. D. H. G. Tuttle, M. D. T. H. Willard, M. D. |
| Michigan Mutual Life, Detroit, Mich. | W. G. Hutchinson, M. D. |
| Minnesota Mutual Life, St. Paul, Minn. | C. N. McCloud, M. D. |
| Missouri State Life, St. Louis, Mo. | B. Y. Jaudon, M. D. |

Companies and Representatives 363

| | |
|--|---|
| Mutual Benefit Life, Newark, N. J. | { C. D. Bennett, M. D. C. P. Clark, M. D. Archibald Mercer, M. D. W. A. Reiter, M. D. G. A. Van Wagenen, M. D. W. R. Ward, M. D. |
| Mutual Life Assurance Co., Waterloo, Ont., Can. | { J. F. Honsberger, M. D. J. M. Livingston, M. D. |
| Mutual Life of New York, New York, N. Y. | { E. McP. Armstrong, M. D. W. M. Bradshaw, M. D. F. H. Carber, M. D. W. S. Gardner, M. D. Lefferts Hutton, M. D. W. E. Porter, M. D. E. F. Russell, M. D. H. H. Schroeder, M. D. F. S. Weisse, M. D. R. L. Willis, M. D. |
| Mutual Trust Life, Chicago, Ill. | W. A. Peterson, M. D. |
| National Life of U. S. A., Chicago, Ill. | W. A. Jaquith, M. D. |
| National Life, Montpelier, Vt. | { A. B. Bisbee, M. D. E. A. Colton, M. D. |
| New England Life, Boston, Mass. | { D. N. Blakely, M. D. E. W. Dwight, M. D. J. M. Little, M. D. W. B. Robbins, M. D. |

Thirty-Fifth Annual Meeting

New York Life,
New York, N. Y.

T. W. Bickerton, M. D.
R. A. Fraser, M. D.
W. J. Hamer, M. D.
C. L. Harrison, M. D.
A. B. Hobbs, M. D.
M. L. King, M. D.
E. H. Lines, M. D.
Paul Mazzuri, M. D.
J. H. North, M. D.
O. H. Rogers, M. D.
P. E. Tiemann, M. D.
H. P. Woley, M. D.

N. American Life Assur. Co.,
Toronto, Ont., Can.

T. D. Archibald, M. D.

North American Life,
Chicago, Ill.

C. B. Irwin, M. D.

Northwestern Mutual Life,
Milwaukee, Wis.

J. W. Fisher, M. D.
G. A. Harlow, M. D.
L. C. Sykes, M. D.
William Thorndike, M. D.
D. E. W. Wenstrand, M. D.

Northwestern National Life,
Minneapolis, Minn.

H. W. Cook, M. D.

Occidental Life,
Los Angeles, Calif.

{ Dudley Fulton, M. D.
D. W. Skeel, M. D.

Pacific Mutual Life,
Los Angeles, Calif.

W. W. Beckett, M. D.

Pan-American Life,
New Orleans, La.

Marion Souchon, M. D.

Companies and Representatives 365

Penn Mutual Life,
Philadelphia, Pa.

J. P. Chapman, M. D.
H. K. Dillard, M. D.
J. U. Hobach, M. D.
J. P. Hutchinson, M. D.
Harry Toulmin, M. D.

Peoria Life,
Peoria, Ill.

G. W. Parker, M. D.

Philadelphia Life,
Philadelphia, Pa.

S. W. Gadd, M. D.

Phoenix Mutual Life,
Hartford, Conn.

A. H. Griswold, M. D.
W. D. Morgan, M. D.
R. L. Rowley, M. D.

Provident Life & Trust Co.,
Philadelphia, Pa.

Herbert Old, M. D.
C. H. Willits, M. D.

Prudential Ins. Co. of Amer.,
Newark, N. J.

C. T. Brown, M. D.
E. G. Dewis, M. D.
W. G. Exton, M. D.
Paul Fitzgerald, M. D.
F. I. Ganot, M. D.
G. E. Kanouse, M. D.
W. P. Lamb, M. D.
L. F. MacKenzie, M. D.
J. A. Patton, M. D.
J. E. Pollard, M. D.
M. K. Smith, M. D.
W. P. Watson, M. D.

Reliance Life,
Pittsburgh, Pa.

O. M. Eakins, M. D.
W. W. Hobson, M. D.
A. A. Wagner, M. D.

366 **Thirty-Fifth Annual Meeting**

| | |
|---|---|
| Reserve Loan Life, Indianapolis, Ind. | F. L. Truitt, M. D. |
| Royal Union Mutual Life, Des Moines, Iowa | J. T. Priestley, M. D. |
| Security Mutual Life, Binghamton, N. Y. | { W. B. Aten, M. D. R. L. Lounsherry, M. D. |
| Standard Life Assur. Co., Montreal, Que., Can. | C. F. Martin, M. D. |
| State Life, Indianapolis, Ind. | { C. R. Henry, M. D. J. L. Larway, M. D. C. B. McCulloch, M. D. |
| State Mutual Life, Worcester, Mass. | { E. B. Bigelow, M. D. Homer Gage, M. D. C. D. Wheeler, M. D. |
| Sun Life, Montreal, Que., Can. | { C. C. Birchard, M. D. W. F. Hamilton, M. D. |
| Southern Life and Trust Co., Greensboro, N. C. | H. F. Starr, M. D. |
| Southern States Life, Atlanta, Ga. | C. E. Waits, M. D. |
| Southland Life, Dallas, Tex. | { J. T. Montgomery, M. D. J. S. Turner, M. D. |
| Southwestern Life, Dallas, Tex. | Whitfield Harral, M. D. |

Companies and Representatives 367

Travelers,
Hartford, Conn.

{ J. T. Cabaniss, M. D.
W. W. Dinsmore, M. D.
P. G. Drake, M. D.
L. C. Grau, M. D.
F. L. Grosvenor, M. D.
Euen VanKleeck, M. D.
McL. C. Wilson, M. D.

Union Central Life,
Cincinnati, Ohio

{ Charles Maertz, M. D.
William Muhlberg, M. D.
W. O. Pauli, M. D.

Union Mutual Life,
Portland, Me.

E. M. Northcott, M. D.

United States Life,
New York, N. Y.

{ J. P. Munn, M. D.
H. A. Pardee, M. D.

Volunteer State,
Chattanooga, Tenn.

J. B. Steele, M. D.

West Coast Life,
San Francisco, Calif.

M. O. Austin, M. D.

DELEGATES FROM ASSOCIATE MEMBER COMPANIES

John W. Abbott, M. D., Maryland Assurance Corporation,
Baltimore, Md.

W. F. Blackford, M. D., Commonwealth Life Insurance Com-
pany, Louisville, Ky.

G. E. Crawford, M. D., Cedar Rapids Life Insurance Com-
pany, Cedar Rapids, Iowa.

Robert J. Graves, M. D., United Life and Accident Insurance
Co., Concord, N. H.

M. M. Lairy, M. D., Lafayette Life Insurance Company,
Lafayette, Ind.

John R. Neal, M. D., Mutual Life of Illinois, Springfield, Ill.

C. E. Schilling, M. D., Ohio State Life Insurance Co., Colum-
bus, Ohio.

S. C. Stanton, M. D., Farmers National Life Insurance Co.
of America, Chicago, Ill.

Carl Stutsman, M. D., Merchants Life Ins. Co., Des Moines,
Iowa.

DECEASED MEMBERS

| | |
|-----------------------------|---------------------|
| A. W. Barrows, M. D. | Hartford, Conn. |
| Charles Bernacki, M. D. | New York, N. Y. |
| William R. Bross, M. D. | New York, N. Y. |
| Chauncey R. Burr, M. D. | New York, N. Y. |
| Robert L. Burrage, M. D. | Newark, N. J. |
| James Campbell, M. D. | Hartford, Conn. |
| Frederick W. Chapin, M. D. | Springfield, Mass. |
| Ferdinand E. Chatard, M. D. | Baltimore, Md. |
| Thomas C. Craig, M. D. | New York, N. Y. |
| Edward Curtis, M. D. | New York, N. Y. |
| Clark W. Davis, M. D. | Cincinnati, Ohio |
| William B. Davis, M. D. | Cincinnati, Ohio |
| Charles A. Devendorf, M. D. | Detroit, Mich. |
| Frank Donaldson, M. D. | Baltimore, Md. |
| Z. Taylor Emery, M. D. | New York, N. Y. |
| Thomas A. Foster, M. D. | Portland, Me. |
| Thomas H. Gage, M. D. | Worcester, Mass. |
| Walter R. Gillette, M. D. | New York, N. Y. |
| Frank S. Grant, M. D. | New York, N. Y. |
| Landon Carter Gray, M. D. | New York, N. Y. |
| Ignatius Haines, M. D. | Boston, Mass. |
| Edward H. Hamill, M. D. | Newark, N. J. |
| William W. Hitchcock, M. D. | Los Angeles, Calif. |
| Edgar Holden, M. D. | Newark, N. J. |
| John Homans, M. D. | Boston, Mass. |
| John Homans, 2d, M. D. | Boston, Mass. |
| Abel Huntington, M. D. | New York, N. Y. |
| Arthur Jukes Johnson, M. D. | Toronto, Ont. |
| John M. Keating, M. D. | Philadelphia, Pa. |
| William W. Knight, M. D. | Hartford, Conn. |
| Edward Lambert, M. D. | New York, N. Y. |

| | |
|-------------------------------|--------------------|
| John B. Lewis, M. D. | Hartford, Conn. |
| Henry P. Lyster, M. D. | Detroit, Mich. |
| Lewis McKnight, M. D. | Milwaukee, Wis. |
| Elias J. Marsh, M. D. | Paterson, N. J. |
| Allison Maxwell, M. D. | Indianapolis, Ind. |
| Francis D. Merchant, M. D. | New York, N. Y. |
| William R. Miller, M. D. | Hartford, Conn. |
| William Natress, M. D. | Toronto, Ont. |
| Frank K. Paddock, M. D. | Pittsfield, Mass. |
| Oliver P. Rex, M. D. | Philadelphia, Pa. |
| James F. W. Ross, M. D. | Toronto, Ont. |
| Gurdon W. Russell, M. D. | Hartford, Conn. |
| George R. Shepherd, M. D. | Hartford, Conn. |
| Melancthon Storrs, M. D. | Hartford, Conn. |
| Brandreth Symonds, M. D. | New York, N. Y. |
| H. Cabell Tabb, M. D. | Richmond, Va. |
| James Thorburn, M. D. | Toronto, Ont. |
| James D. Thorburn, M. D. | Toronto, Ont. |
| Henry Tuck, M. D. | New York, N. Y. |
| S. Oakley Van der Poel, M. D. | New York, N. Y. |
| A. L. Vanderwater, M. D. | New York, N. Y. |
| Clinton D. W. VanDyck, M. D. | New York, N. Y. |
| A. C. Ward, M. D. | Newark, N. J. |
| Joseph H. Webb, M. D. | Waterloo, Ont. |
| Frank Wells, M. D. | Boston, Mass. |
| Franklin C. Wells, M. D. | New York, N. Y. |
| George W. Wells, M. D. | New York, N. Y. |
| A. H. Whitridge, M. D. | Baltimore, Md. |
| George Wilkins, M. D. | Montreal, Que. |
| G. S. Winston, M. D. | New York, N. Y. |
| Albert Wood, M. D. | Worcester, Mass. |
| Green V. Woollen, M. D. | Indianapolis, Ind. |
| Joseph C. Young, M. D. | Newark, N. J. |

INDEX

Address of the President, Dr. William R. Ward, 16
Address of Welcome, Dr. John R. Hardin, President of The Mutual Benefit Life Insurance Company, 5
Annual Dinner, 340
Assessment, Report of Committee on, Dr. C. L. Christiernin, Chairman, 27

Bailey, Dr. W. C., Report on Diastolic Blood Pressure, 143
Blood Pressure, Some Practical Observation on the Taking of, By Dr. G. E. Crawford, 38

Carber, Dr. Frank, The Present Status of Goiter, 270
Clark, Dr. Charles P., Demonstration of Urinary Tests, 250
Committee on Dreyer Measurements in Relation to Life Insurance Underwriting Practice, Dr. Wm. Muhlberg, Chairman. Discussion by Dr. W. O. Pauli and Dr. Dwight, 28
Committee on Urinary Impairments, Report of, By Dr. J. Allen Patton, 244
Discussion by Drs. Cook, Rockwell, Bradshaw, Rogers and Knight, 253
Companies and Their Representatives, 359
Craig, Mr. James D., Disability Benefit in Life Policies, 294
Crawford, Dr. G. E., Some Practical Observations on the Taking of Blood Pressure, 38

Deceased Members, 368
Demonstration of Urinary Tests, By Dr. Charles P. Clark and Dr. F. B. Kingsbury. Discussion by Drs. Cook, Rockwell, Bradshaw, Benedict and Folin, 250
Diastolic Blood Pressure, By Dr. W. C. Bailey, 143

Disability Benefit in Life Policies, Mr. James D. Craig, Discussion by Drs. Beckett, Grosvenor, Root and Snow, 294

Dreyer Measurements in Relation to Life Insurance Underwriting Practice, Committee on, Dr. Wm. Muhlberg, Chairman, 28

Election of New Members, 3

Election of Officers, 142

Glycosuria, A Proposed Method of Selecting Risks Among Individuals with Occasional Slight, By Dr. F. H. McCrudden, 156
Discussion by Drs. Benedict, Rolph, Bradshaw, Dwight, Folin and Rockwell, 229

Goiter, The Present Status of, By Dr. Frank Carber, 270
Discussions by Drs. Hutchinson, Bradshaw, Pollard and Russell, 281

Hardin, Mr. John R., Address of Welcome, 5

Hart, Dr. T. Stuart, The Significance of Heart Murmurs and Irregularities, 107

Health Examinations for Policyholders, The Value of Periodic, Dr. Harry Toulmin, 47
Discussion by Drs. Knight, Pauli, Geiringer, Fisk and Honsberger, 51

Heart Murmurs and Irregularities, The Significance of, Dr. T. Stuart Hart, 107
Discussion by Drs. Root, Hart, Sykes, Baker, McCrudden and Mackenzie, 120

Huston, Dr. Ross, Practical Methods for Promoting the Consideration of Applications by Lay Approvers, 145

Hunter, Arthur, and Dr. Oscar H. Rogers, Mortality Study of Impaired Lives, 96

Impaired Lives, Mortality Study of, Dr. Oscar H. Rogers and Arthur Hunter, 96

Kingsbury, Dr. F. B., Demonstration of Urinary Tests, 252

McCradden, Dr. Francis H., A Proposed Method of Selecting Risks Among Individuals with Occasional Slight Glycosuria, 156

Members of the Association of Life Insurance Medical Directors, List of, 347

Miller, Dr. James Alexander, Relative Value of the Various Factors in the Diagnosis of Suspected Tuberculosis, 127

Mortality Study of Impaired Lives, Dr. Oscar H. Rogers and Arthur Hunter, 96

Obituary, Dr. Chauncey Rae Burr, 7

Obituary, Dr. Z. Taylor Emery, 9

Obituary, Dr. Ignatius Haines, 8

Obituary, Dr. William Ward Knight, 11

Obituary, Dr. Brandreth Symonds, 12

Obituary, Dr. Franklin C. Wells, 14

Practical Methods for Promoting the Consideration of Applications by Lay Approvers, Dr. Ross Huston, 145
Discussion by Dr. Kanouse and Mr. Everett, 152

Reminiscences and After Dinner Talk, Dr. George A. Van Wagner, 342

Rhodes, Mr. E. E., Some Non-Medical Thoughts Regarding Selection, 262

Rogers, Dr. Oscar H. and Arthur Hunter, Mortality Study of Impaired Lives, 96

Selection, Some Non-Medical Thoughts Regarding, Mr. E. E. Rhodes, 262

Toulmin, Dr. Harry, The Value of Periodic Health Examinations, 47

Tuberculosis, Relative Value of the Various Factors in the Diagnosis of Suspected, Dr. James Alexander Miller, 127

Urinary Impairments, Report of Committee on, Dr. J. Allen
Patton, 244

Demonstration of Urinary Tests by Drs. Clark and
Kingsbury, 250

Discussions, Drs. Cook, Rockwell, Bradshaw, Benedict
and Folin, 253

Van Wagner, Dr. George A., Reminiscences and After Dinner
Talk, 342

Ward, Dr. William R., Address of the President, 16

